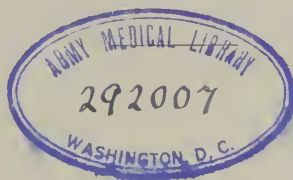


A horizontal section of the eye. (Aller.)

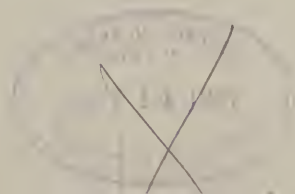
NOTES
ON
THE EYE,
FOR THE USE OF
STUDENTS,

BY
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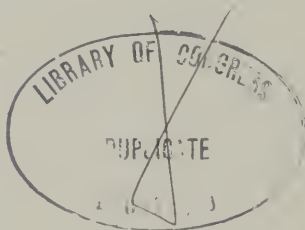


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PREFACE.

The object of this little handbook is to help students by giving them the elements of ophthalmology in a condensed form. It is not my desire to minimize medical education but to increase the knowledge of the graduate by simplifying the method of his instruction. The short time allotted to the eye in our medical schools and the large size of the text books on the subject are so disproportioned as to cause confusion and despair to the student. The result is abandonment of the text book and reliance upon the notes taken in the lecture room. This little book, which contains nothing original, but has been compiled from standard works, is not intended to take the place of the large size text book, at least one of which every student and practitioner should have, but is designed to supplant the inaccurate notes taken by the student in the lecture room and to suppress a practice so disagreeable to the teacher and injurious to the pupil. It is expected that each subject will be explained in detail in the class room, these notes serving only as a frame work around which to build a course of lectures. Only the gross anatomy of the eye has been given, only an outline of refraction, and where it was possible to include under one description several forms of a disease it has been done, as I recognize that there is enough matter to fill a medical course to overflowing which is of more importance to the student than the coats of the retina, or the formula for calculating the index of refraction of a transparent medium. I have also slighted those diseases which have to be diagnosed with the ophthalmoscope as I doubt the diagnostic value of an ophthalmoscope in the hands of the average general practitioner. If it is conceded that

these notes are more accurate, more comprehensive and more useful than the students' class room notes, I have accomplished my full purpose in writing them.

511 North Garrison Avenue.

September 1897.

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CHAPTER I.

ANATOMY OF THE EYE.

THE ORBITS.

These four-sided, pyramidal cavities, a little over an inch and a half deep, are formed by seven bones, frontal, sphenoid, ethmoid, superior maxillary, palate, malar and lachrymal. The bones are covered by periosteum and the orbital space not filled by the globe, optic nerve, muscles and vessels is occupied by fat and connective tissue. This

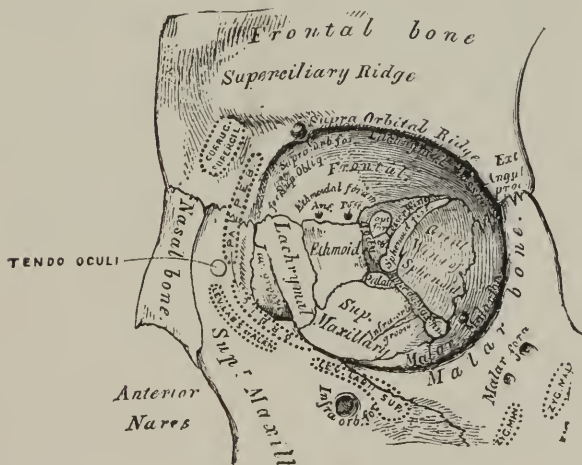


FIG. 1.—Left orbit. (Gray.)

connective tissue becomes thickened in parts so as to form sheaths for the muscles; it also forms a fascia which surrounds the optic nerve and then spreads over the eyeball, making a smooth socket in which it rotates. Anteriorly this fascia is called Tenon's capsule, posteriorly Bonnet's capsule. On the inner wall of the orbit is the groove in

which is lodged the lachrymal sac. In front of this groove is the insertion of the orbicularis, the muscle which closes the eye lids, and behind the groove is the insertion of the tensor tarsi muscle which holds the lids close to the globe. In the angle formed by the roof of the orbit and the nasal wall, and a short distance behind the orbital rim, is the loop or pulley through which passes the tendon of the su-

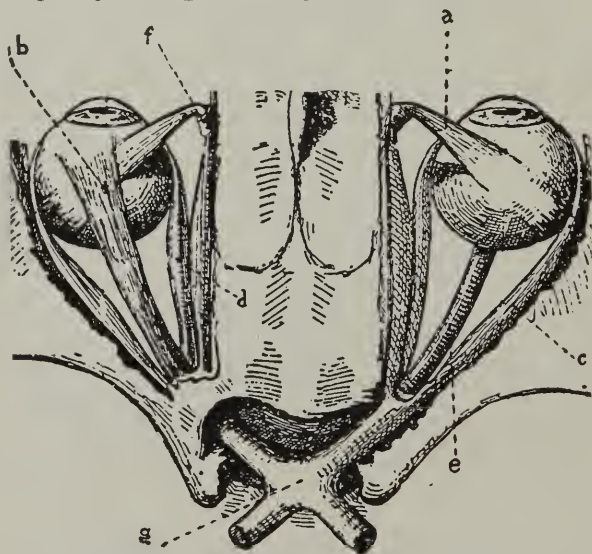


FIG. 2.—The ocular muscles seen from above. a, Superior oblique; b, Superior rectus; c, External rectus; d, Internal rectus; e, Optic nerve; f, Pulley of superior oblique; g, Optic commissure.

perior oblique muscle. In the angle formed by the roof and the temporal wall and just under the edge of the orbit is a fossa which holds the lachrymal gland. In the apex of the orbit is the sphenoidal fissure, which transmits the third, fourth and sixth nerves, the ophthalmic division of the fifth nerve and the ophthalmic vein. Above the inner end of the sphenoidal fissure is the optic foramen, a cylindrical canal which transmits the optic nerve and ophthalmic artery. The ophthalmic artery, a branch of the internal carotid, supplies blood to the orbit and its contents.

THE OCULAR MUSCLES.

The levator palpebrae superioris muscle, which lifts the upper lid, the superior oblique and the four recti (internal, external, inferior and superior) muscles, all arise at the apex of the orbit around the optic foramen. Though the superior oblique arises at the apex of the orbit, the direction of its force is changed by passing through the pulley, before mentioned, which is situated in the angle formed by the roof and the nasal wall of the orbit. It passes backward and outward from this pulley to its insertion in the sclera. The inferior oblique muscle arises from the floor of the orbit near the lachrymal groove and passes directly backward and outward to its attachment in the sclera. The

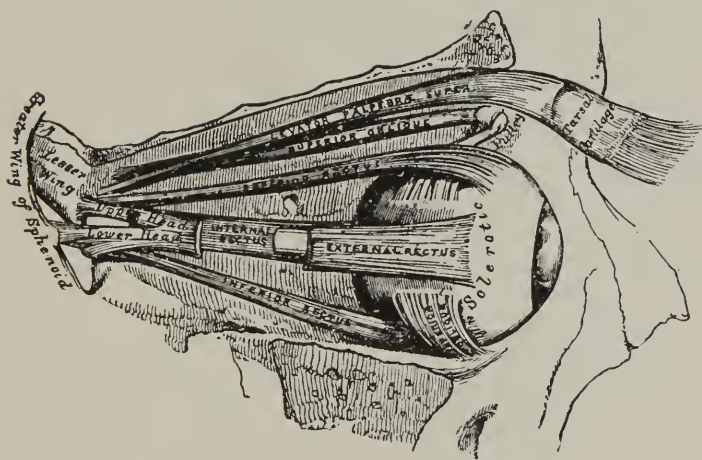


FIG. 3.—Muscles of the right eye. (Gray.)

motor muscles of the eye are supplied with blood by the muscular branches from the ophthalmic artery. The external rectus muscle is supplied by the sixth nerve, the superior oblique by the fourth nerve and the four remaining motor muscles, as well as the levator palpebrae superioris, by the third nerve.

THE LIDS.

Under the skin of the lids is a thin layer of connective tissue, and under this the fibres of the orbicularis muscle. Under the orbicularis are the tarsi, formerly called carti-

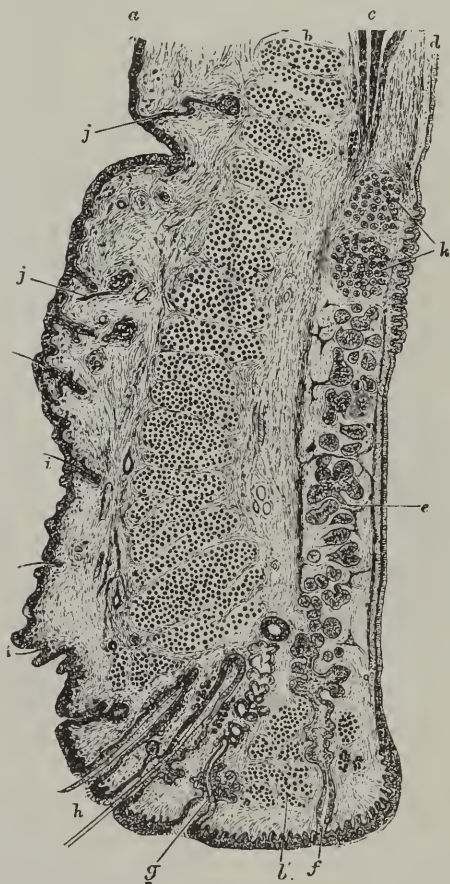


FIG. 4.—Vertical section through the upper eye lid. (Schafer.) a, Skin; b, Orbicularis; b', Ciliary bundle; c, Involuntary muscle of eye lid; d, Conjunctiva; e, Tarsus; f, Meibomian gland; g, Sebaceous gland, or gland of Moll; h, Eye lashes; i, Small hairs in skin; j, Sweat gland; k, Glands of Krause.

lage, now known to be dense fibrous tissue. There is one of these thin, flat, elongated plates in each lid to give it

form and support, the tarsus of the upper lid being twice as wide as the tarsus of the lower. The tarsi are connected at their extremities and also bound to the subjacent bone by the internal and external palpebral ligaments. Under



FIG. 5.—The tarsi. (Schwalbe.)

the tarsi and in grooves in their substance are the meibomian glands. They number about thirty in the upper and twenty in the lower lid. They resemble currants on a stem, the stem lying across the tarsus and at right angles to the edge of the lid. Their ducts open on the free margin of the lids. They are like sebaceous glands and the

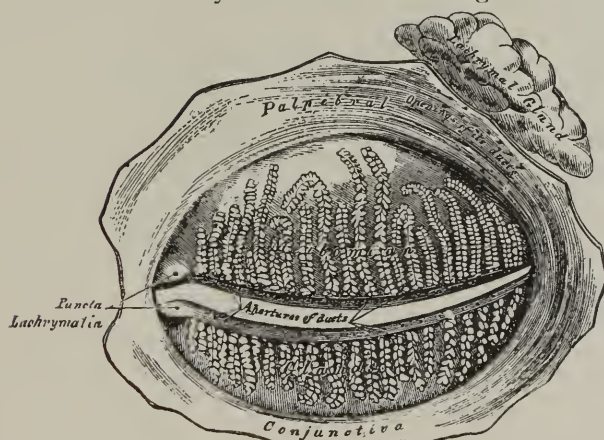


FIG. 6.—Position of meibomian glands, the conjunctiva removed. (Gray.)

fluid they secrete prevents adhesion of the lid borders. Under the meibomian glands is the conjunctiva, the mucous membrane which covers the inner surface of the lids. The opening between the lids is called the palpebral fissure. The nasal end of the fissure is the inner canthus, the temporal end is the outer canthus. The eye lashes are ar-

ranged in two rows and their follicles are supplied with sebaceous glands: the glands of Moll. The lids are closed by the orbicularis muscles which is supplied by the facial nerve or portio dura of the seventh nerve, and are opened by the levator palpebrae superioris which is supplied by the third nerve.

THE LACHRYMAL APPARATUS.

The lachrymal apparatus is divided into the secretory part and the excretory part. The former is composed of the lachrymal gland and a series of small glands called the glands of Krause, which lie just under the palpebral conjunctiva near the fornix. They are much more numerous in the upper than in the lower lid. The lachrymal gland

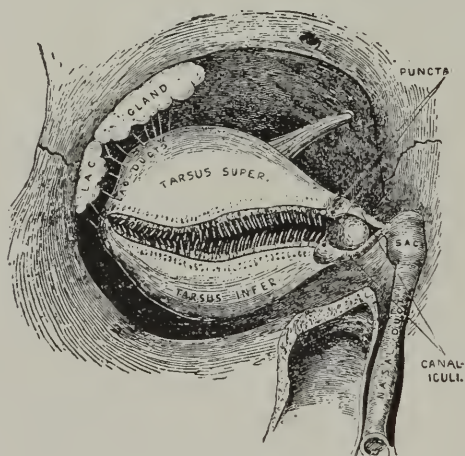


FIG. 7.—The Lachrymal apparatus. (Juler.)

is lodged in a fossa at the upper and outer angle of the orbit. It is about the size of the kernel of an almond, and is composed of a large and small lobe united by a fascia. It secretes tears, which empty into the conjunctival sac at the superior fornix, through a number of small tubes; these open by about twelve orifices near the temporal side. The excretory apparatus begins as minute openings: the

puncta, which are located on the lid border about a quarter of an inch from the inner canthus. The puncta open into small tubes, the canaliculi, which empty by a common orifice into the side of the lachrymal sac. The sac is continuous below with the lachrymo-nasal duct, which empties into the inferior nasal fossa. The total length of the sac and duct is about one inch.

THE CONJUNCTIVA.

The conjunctiva is a mucous membrane. It presents two portions. The tarsal portion lines the eye lids. The ocular portion covers all of the visible front of the eye ball except the cornea. The two parts are connected by a loop or fold of mucous membrane called the fornix or sinus. In the fornix are located the accessory lachrymal glands: the glands of Krause. On the conjunctiva near the inner canthus is a round, fleshy body, an accumulation of glandular follicles called the caruncle, and between the caruncle and the edge of the cornea is the plica semilunaris, a fold of conjunctiva which is the analogue of the third eye lid of some animals. The tarsal conjunctiva is slightly velvety, due to numerous fine grooves and pits in its surface; the elevations between the grooves are called papillae. Under the papillae is a layer of fibrous tissue which is closely adherent to the tarsus, and in this fibrous layer are found numerous lymphoid follicles. The blood supply of the conjunctiva is from branches of the ophthalmic, facial and internal maxillary arteries. The nerve supply is very profuse and comes from the fifth.

THE CORNEA.

The cornea is the transparent, glassy looking, circular membrane which forms part of the front of the eye ball. It has no blood vessels, its nutrition being supplied by lymph from a loop of vessels that encircles its periphery. The nerve supply is from the fifth and is very profuse. The cornea has five layers:—

1. In front a layer of epithelium, six to eight cells deep, which is continuous with the epithelium of the conjunctiva.

2. Bowman's membrane, a dense, firm, homogeneous fibrous tissue.

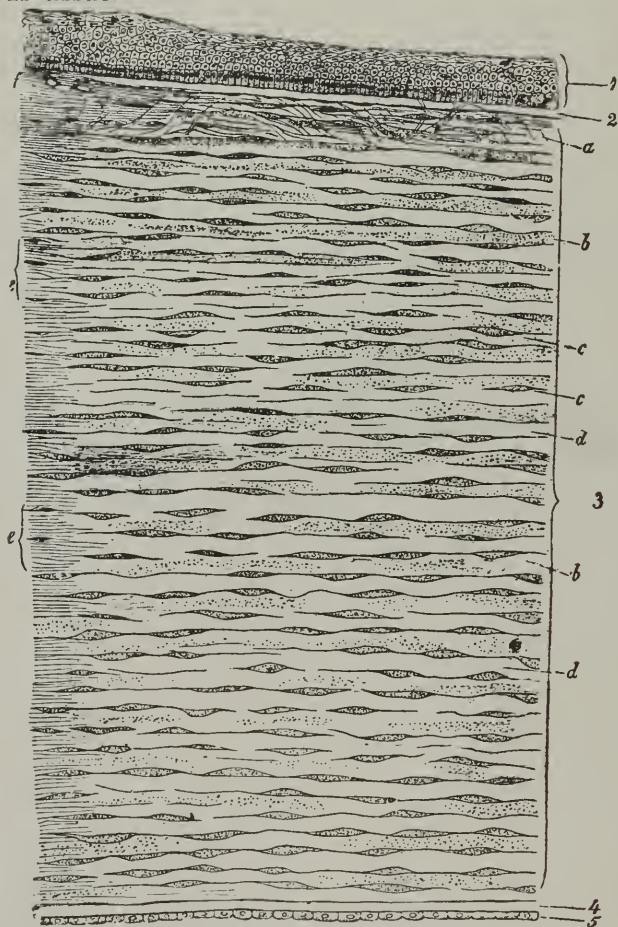


FIG. 8.—Section of cornea near the limbus. (Schafer.) 1. Epithelium; 2. Bowman's membrane; 3. Substantia propria; 4. Descemet's membrane; 5. Endothelium; a, Oblique fibres in the anterior layer of the substantia propria; b, Lamellae the fibres of which are cut across, producing a dotted appearance; c, Corneal corpuscles appearing fusiform in section; d, Lamellae the fibres of which are cut longitudinally; e, Transition to the sclerotic, with more distinct fibrillation, and surmounted by a thicker epithelium; f, Small blood vessel cut across near the margin of the cornea.

3. Substantia propria or proper substance of the cornea, a transparent fibrous tissue, not as dense or resistant as the preceding, forming the greater part of the thickness of the cornea. It is composed of about sixty layers, the fibres in each running in the opposite direction to the fibres in the layer above and below. Between these layers are found cell spaces which communicate with each other and serve as lymph channels. Within the cell spaces are found the corpuscles, the connective tissue cells of the cornea.

4. Descemet's membrane, a thin, elastic, brittle, fibrous layer, the most resisting of the cornea.

5. A single layer of endothelial cells.

THE SCLERA.

The sclera composes all the outer tunic of the eye ball except that part represented by the cornea. It is also fibrous tissue and is tough, resistant and slightly elastic. The part called the white of the eye is a portion of the sclera and is covered by the ocular conjunctiva. Between the conjunctiva and the sclera is a loose connective tissue called the episclera. Behind, and a little to the nasal side, the optic nerve pierces the sclera. It does not enter in one bundle but divides and enters through numerous openings. This seive-like part of the sclera is called the lamina cribrosa. Around the lamina cribrosa numerous blood vessels and nerves pierce the sclera.

THE IRIS.

From the point of union between the sclera and cornea a curtain is suspended in the eye. This curtain is composed of muscular fibres, pigment, blood vessels, nerves and connective tissue, and is the iris. In the center of the iris is a round opening: the pupil. The muscular fibres of the iris are involuntary (unstriped) and are divided into radiating and circular fibres. The latter are arranged around the pupil and act as a sphincter in contracting it. These fibres are controlled by the third nerve. The radiating fibres are supposed to dilate the pupil and are controlled by

the sympathetic nerve. The function of the iris is to regulate the amount of light entering the eye. In accomplishing this its action is reflex, the afferent nerve being the optic, the efferent nerve the third. In front of the iris and between it and the cornea is the anterior chamber. Behind the iris is a triangular space; the posterior chamber. The base of the triangle is the ciliary body; the two sides, the iris and lens; the apex, the point where the pupillary margin of the iris comes in contact with the lens. The anterior and posterior chambers form the aqueous chamber. The long ciliary arteries, two in number, arise from the ophthalmic and pierce the sclera, one on each side of the optic nerve, pass forward between the choroid and sclera to the periphery of the iris and there make the *Circulus Arteriosus Iridis Major*; from which branches run, like the spokes of a wheel, toward the pupil and around its margin form the *Circulus Arteriosus Iridis Minor*.

THE CILIARY BODY.

The ciliary body lies between the iris and the anterior end of the retina. It is firmly adherent to the sclera at the anterior end but loosely attached behind. It is divided into two parts:—

1. The vascular part, which is composed of convoluted blood vessels and pigment, lies next to the vitreous and supplies it and the lens with much of their nourishment. The anterior part of the vascular portion is thrown into seventy or eighty projecting tips called the ciliary processes, from the posterior surface of which passes a transparent ligament, the zonula of Zinn or the suspensory ligament of the crystalline lens.

2. The muscular part, which lies next to the sclera and is the agent of accommodation. Its fibres are unstriated and are arranged in two sets, one parallel with, and the other at right angles to the sclera. The ciliary muscle was formerly called the ciliary ligament. Contraction of the ciliary muscle is produced by those fibres of the ciliary

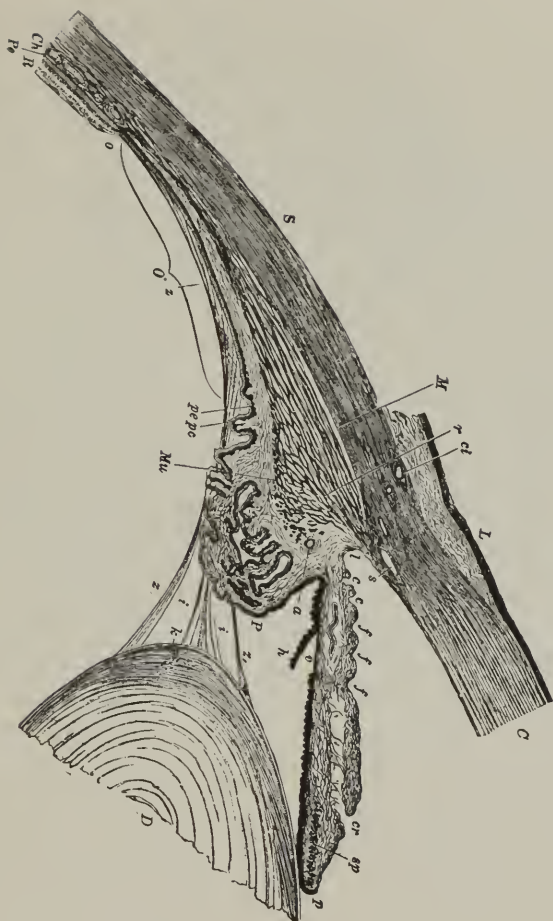


FIG. 9.—Section through the ciliary region. (Fuchs) C, cornea; S, sclera; Ch, choroid; R, retina; Pe, its pigmented epithelium; O, orra serrata; O, pars ciliaris retinae; this is continued over the ciliary processes; pe, pc, pigmented and non-pigmented cells of pars ciliaris; D, lens; M, ciliary muscle; r, its radiating fibres; ci, anterior ciliary artery; S, canal of Schlemm; z, origin of ciliary muscle; c, c, f, f, folds and depressions in anterior surface of iris; cr, a crevice in this surface (? artificial); sp, sphincter pupillae; p, edge of pupil; P, most prominent part of ciliary process; h, pigment behind iris, detached at v; a, blood vessel; z, zonula of Zinn; z', z', its continuation or the suspensory ligament; i, i, spaces between the fibres of the suspensory ligament; k, capsule of lens.

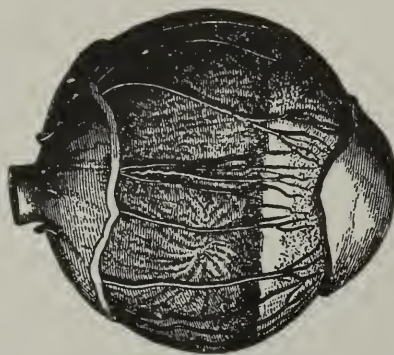


FIG. 10.—The sclera removed, showing the ciliary nerves, passing through the suprachoroidal space to reach the anterior part of the eye, (after Merkel.)

nerves which are derived from the third. The long ciliary nerves, two or three in number are given off from the nasal nerve which is a branch of the ophthalmic; the ophthalmic being the first division of the trigeminus or fifth. The short ciliary nerves, ten or twelve in number arise from the ciliary or lenticular ganglion. This ganglion which is about the size of a pin head is found back of the orbit between the optic nerve and the external rectus muscle. It is supplied by three roots from the fifth, third, and sympathetic nerves. The long and short ciliary nerves pierce the sclera around the optic nerve and pass forward between the choroid and sclera to the ciliary muscle and iris.

THE CHOROID.

Extending from the ciliary body back to the optic nerve and lying next to the sclera is a vascular membrane, the choroid. Its arteries, the twelve to fifteen short posterior ciliary, which arise from the ophthalmic and pass through the sclera around the optic nerve, anastomose freely to form a capillary layer, which helps to nourish the retina and vitreous. Its veins arise from this capillary layer and from the ciliary body and iris and unite into four or six groups. Each group empties near the equator through one vein, the *vena vorticosa*. The *venæ vorticosæ* empty into the oph-

thalmic vein. The iris, ciliary body, and choroid are similar in structure and are called the uveal tract.

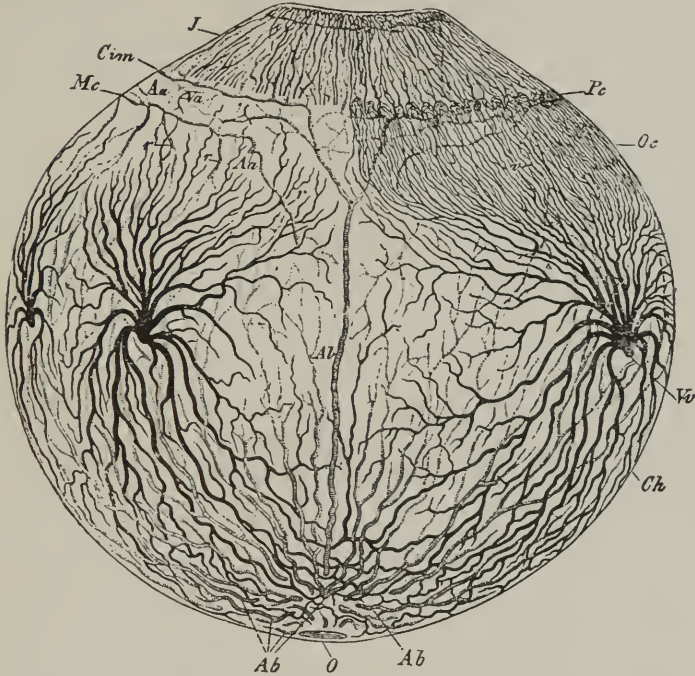


FIG. 11.—Circulation of the choroid. (Leber.) O, optic-nerve entrance; Oc, ciliary region; Pc, ciliary processes; J, iris; Aa, anterior ciliary arteries; Ab, short posterior ciliary arteries; Al, long posterior ciliary; Cim, circulus arteriosus iridis major; Mc, arteries of ciliary muscle; Vv, vena vorticiosa.

THE LENS.

Behind the iris and in contact with the pupillary margin, lies a circular, bi-convex, transparent body composed of albumen and water, the crystalline lens. It is contained in a transparent capsule and is held in position by the suspensory ligament or zone of Zinn, which is composed of delicate connective tissue fibres that arise from the posterior surface of the ciliary processes and are inserted, half in the anterior lens capsule and half in the posterior. The space between these fibres which pass to the front and

back of the lens is called the canal of Petit. The lens is divided into a dense central part: the nucleus and a softer peripheral part: the cortex:

THE VITREOUS.

The interior of the eye ball is filled by a transparent, jelly like substance, the vitreous, which maintains the shape of the eye, and holds the retina and lens in position. It has no blood vessels or nerves and is nourished from surrounding structures. Through its center is a lymph canal, which in foetal life contains an artery. It is surrounded by a hyaloid membrane and is cupped anteriorly to receive the posterior surface of the lens.

THE RETINA.

The fibres of the optic nerve pass through the lamina cribrosa and spread between the choroid and vitreous, forming an almost transparent membrane, the retina, which extends anteriorly to the ciliary body. It is composed of

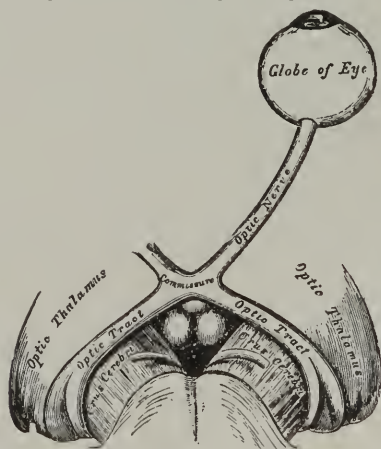


FIG. 12.—Optic tracts and commissure. (Gray.)

ten layers which are made up of nerve structures and modified connective tissue. In the center of the posterior part of the retina is a round, elevated, yellowish spot, one twentieth of an inch in diameter, the macula lutea; and in

the center of the macula is a depression, the fovea centralis, which is the center of direct vision and is the most sensitive part of the retina. The arteria centralis retinae pierces the optic nerve just before it enters the globe and passes into the eye, spreading out in the retina. The retinal veins lie by the side of the arteries.

THE OPTIC NERVES.

The right and left optic tracts pass forward from the brain to unite in the optic groove of the sphenoid bone and form the optic commissure. (Fig. 12.) Within the commissure the nerve fibres undergo partial decussation and emerge in two bundles which are the optic nerves or second cranial nerves. They diverge from each other and passing through the optic foramens, enter the back of the ball a little to the nasal side of its center, the sheath of the nerve

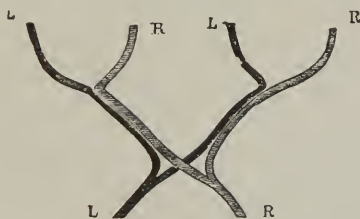


FIG. 13.—Decussation of optic-nerve fibres. (Wells.)

merging into the sclerotic. The decussation in the commissure is of such nature that the fibres of the right tract are distributed to the right side of each retina, and the fibres of the left tract to the left side of each retina. (Fig 13.)

CHAPTER II.

REFRACTION AND PHYSIOLOGY.

REFRACTION.

Refraction is the change which takes place in the direction of rays of light when they pass obliquely from one transparent medium into another of different density.

All transparent solids and liquids are denser than air.

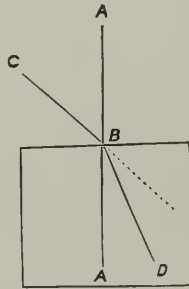


FIG. 14.—A A, perpendicular to surface between air and glass. C B D, ray bent toward perpendicular when passing from rarer medium air into denser medium glass. Reverse the direction and D B C, is a ray bent from the perpendicular when passing from the denser medium glass into the rarer medium air.

A ray of light passing from a rarer into a denser medium is bent toward the perpendicular. A ray of light passing from a denser into a rarer medium is bent from the perpendicular (Fig. 14.)

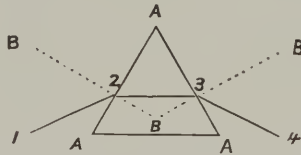


FIG. 15.—A A A, a prism B B B, perpendiculars to sides of prism. 1, 2, 3, ray bent toward perpendicular when passing from air into glass. 2, 3, 4, ray bent from perpendicular when passing from glass into air.

A refracting prism is any transparent body lying between two plane faces which are not parallel. A ray of light

upon entering a prism is bent toward the perpendicular, upon emerging is bent from the perpendicular. A prism always bends rays toward its base (Fig. 15.)

Any transparent medium bounded by two curved surfaces, or one plane and the other curved, is a lens.

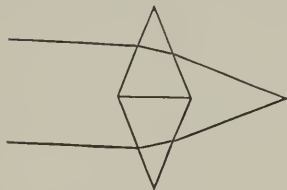


FIG. 16.

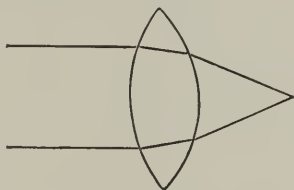


FIG. 17.

A convex lens may be regarded as a series of prisms with their bases directed toward the center (Fig. 16). A convex lens converges parallel rays of light so as to bring them to a focus (Fig. 17).

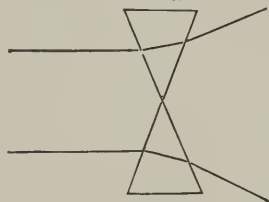


FIG. 18.

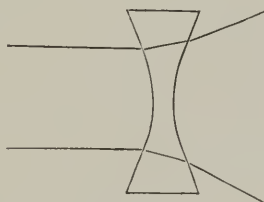


FIG. 19.

A concave lens may be regarded as a series of prisms with their bases directed from the center (Fig. 18). A concave lens diverges parallel rays of light (Fig. 19).

The strength of a lens or its ability to change the direction of rays depends upon the degree of curvature of its surfaces.

Rays of light from distant objects (20 feet or over) enter the eye parallel. Rays of light from near objects (under 20 feet) are divergent when they enter the eye.

In order for an eye to have distinct vision of an object, rays of light from that object must be brought to a focus on the retina. The cornea and crystalline lens act as convex lenses in bringing rays of light which enter the eye to a focus on the retina.

EMMETROPIA.

An eye, which, in a state of rest, brings parallel rays of light to a focus on its retina has normal refraction and is called emmetropic.

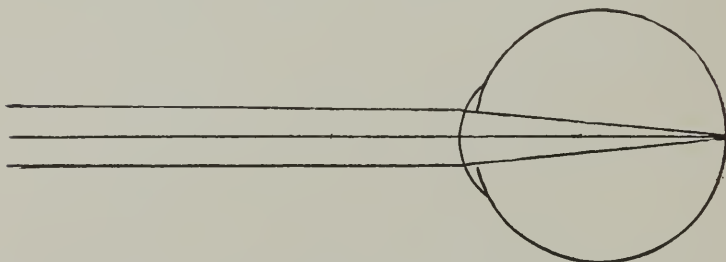


FIG. 20.—Parallel rays of light brought to a focus on the retina as in the emmetropic eye.

ACCOMMODATION.

Rays from a near object are divergent and in order for an emmetropic eye to have distinct vision of a near object it must increase its focal power, as it not only has to focus parallel rays but has first to make the divergence parallel. This is accomplished by contracting the ciliary muscle. Contraction of the ciliary muscle relaxes the suspensory ligament and capsule of the lens. When the pressure of the capsule is relieved the lens becomes more convex by an inherent elasticity. Increase in its focussing power. This power the eye possesses of increasing its focal strength is called accommodation.

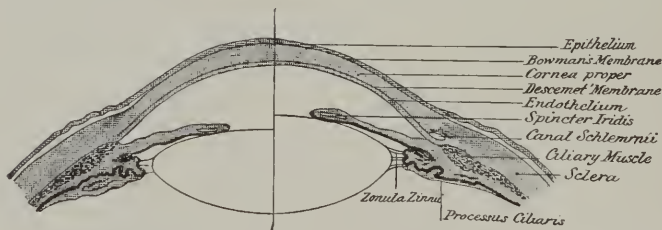


FIG. 21.—The left half represents the eye at rest, the right during accommodation. (Fick.)

PRESBYOPIA.

At about the age of forty-five the lens begin to lose its elasticity, its power of accommmodating is lessened and convex glasses have to be resorted to for near vision. This physiological loss of accommodative power is called presbyopia. As accommodation diminishes the reading glass must be strengthened, necessitating a change about every two years.

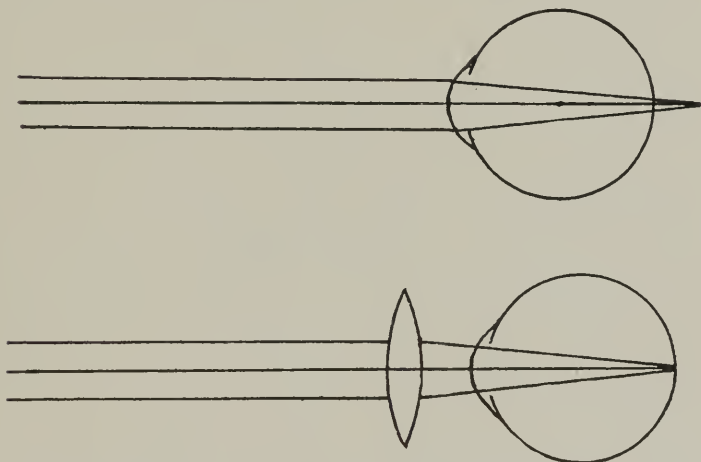


FIG. 22.—The upper figure shows parallel rays of light focussed behind the retina as in hyperopia. The lower figure shows the influence of a convex lens in bringing the focus to the retina.

HYPEROPIA.

If the focus of parallel rays is at an imaginary point behind the retina, the eye being at rest, (i. e. not accommodating) it is far sighted or hyperopic. Hyperopia is due to shortness of the antero-posterior axis of the eye ball or to lack of sufficient focal strength in the cornea and lens. Since by accommodating the lens can increase its focal strength, hyperopic eyes accommodate constantly. This constant exercise of the ciliary muscles produces a condition commonly known as eye strain. Distant objects are seen plainly unless the hyperopia be very pronounced.

Near work is difficult and often painful. We compensate for the lack of focal strength and relieve the ciliary muscle of its strain by the use of convex lenses. (Fig. 22.)

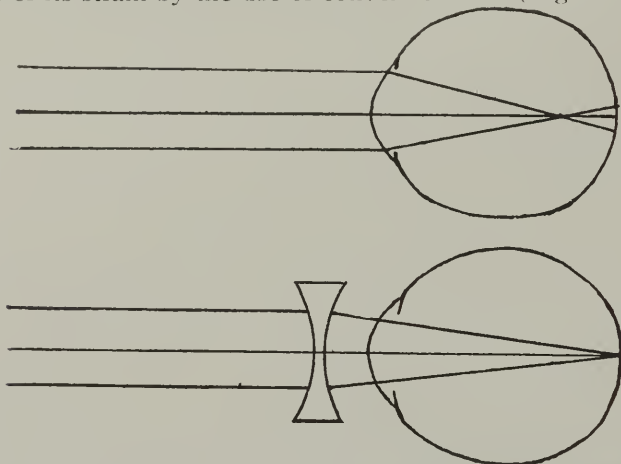


FIG. 23.—The upper figure shows parallel rays of light focussed in front of the retina as in myopia. The lower figure shows the influence of a concave lens in moving the focus back to the retina.

MYOPIA.

If the focus of parallel rays is at a point in front of the retina, the eye being at rest, it is near sighted or myopic. Myopia is due to too great length of the antero-posterior axis of the eye ball, or to too great focal strength of the



FIG. 24.—Ciliary muscle of a hyperopic eye. RF radiating fibres; CF circular fibres.

cornea and lens. Since there is no way of decreasing the focal strength of the lens no effort on the part of a myope

can overcome his defect. Distant objects, are seen poorly and if the myopia is pronounced, are not seen at all. We neutralize the too great strength of the lens and secure good vision by the use of concave lenses. (Fig. 23.)

In hyperopia the effort of accommodation necessitates the constant exercise of the circular fibres of the ciliary muscle and we find in hyperopic eyes that the circular fibres are increased in size and number as in figure 24.

Accommodation would make the vision of a myopic eye worse and we find in these eyes that the circular fibres of the ciliary muscle are small in size and number as in figure 25.



FIG. 25.—Ciliary muscle of a myopic eye.

ASTIGMATISM.

The axis of the eye is a line drawn from the center of the cornea, through the center of the ball, to a point between the optic nerve and macula lutea. The corneal end of this line is the anterior pole, the other end, the posterior pole of the eye. A circle around the ball at an equal distance from the poles is the equator. Other circles around the ball, passing through the poles, are meridians.



FIG. 26.—Cross lines as seen by an emmetropic eye and by two astigmatic eyes. (Juler.)

In emmetropia, hyperopia and myopia the curvature of the cornea is the same in every meridian, and its refract-

ing power is the same through every part, vertical, horizontal and oblique. All rays that enter these eyes are brought to a common focus. Sometimes the cornea has meridians of unequal curvature producing greater refraction in some meridians than in others. Such a condition constitutes astigmatism. In astigmatism the rays passing

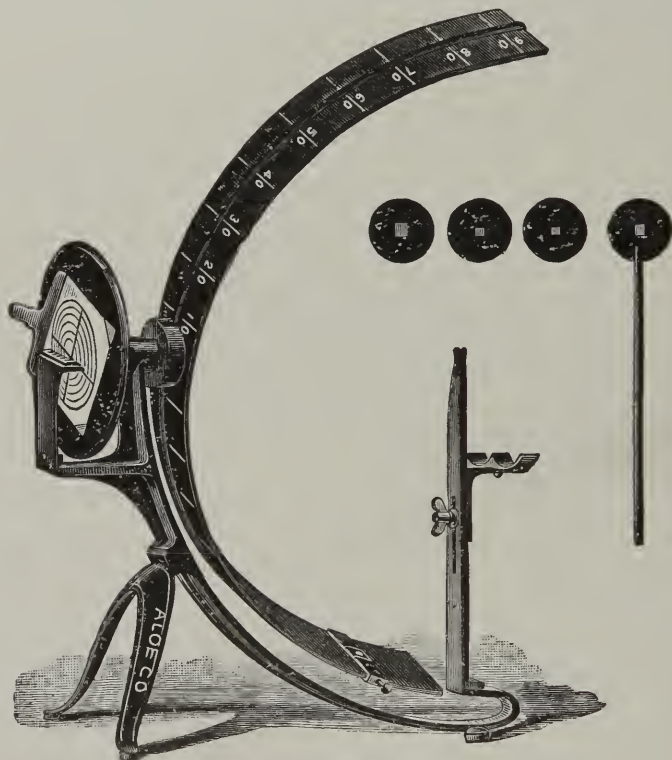


FIG. 27.—A perimeter, used in measuring the extent of the field of vision.

through the meridian of greatest refraction reach their focus nearest the cornea, while those passing through the least refracting meridian come to a focus farthest back. The meridians of highest and lowest refracting power are at right angles to each other and are called the principal meridians.

CONVERGENCE.

When you look straight at any object your eyes are said to fix it, in other words, when an eye is directed toward anything so that the image of the thing looked at falls upon the fovea centralis of the eye, the eye is said to fix that object. Normally both eyes fix the same object, and in order to do this when the object is brought near to the face, both eyes have to turn inward and the nearer the object is, the more the eyes turn in. The turning in of the eyes necessary to fix near objects is called convergence.

FIELD OF VISION.

When the eye is fixed on an object, other objects besides the one looked at are visible. Those nearest the one fixed are most distinct and the greater the distance of an object from the one fixed, the less distinctly is it seen. That area in which objects are visible, the eye being fixed, is the field of vision.

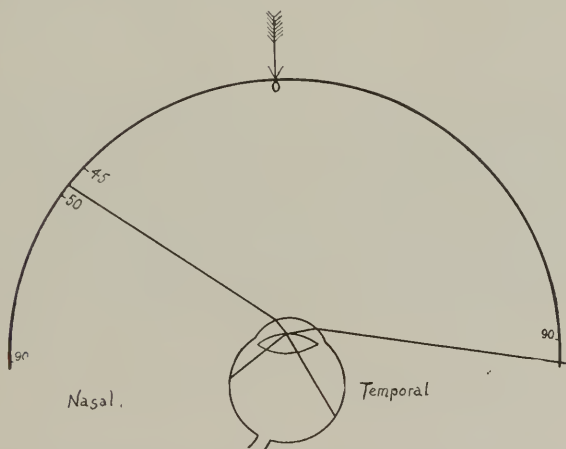


FIG. 28.—Field of vision of a right eye. The arrow at *o* being fixed (looked at) all objects on the temporal side within the area described by about 95 degrees of a circle are visible; all objects on the nasal side within about 48 degrees are visible. The nasal side of the field is restricted by the bridge of the nose.

COLOR PERCEPTION.

Many theories have been offered to explain the phenomenon of color perception, but none has yet supplanted

the Young-Helmholtz. This is that we have three primary color perceptions corresponding to the three primary colors of nature, and that there are red perceptive fibres, green perceptive fibres and violet perceptive fibres in our retinas. These different nerve fibres are stimulated by light waves of different lengths. Equal stimulation of all three produces the sensation of white and just as all the colors in nature can be produced by mixing the spectrum red, green and violet so can every color sensation be produced by stimulation of the red, green and violet perceptive fibres in varying proportions. The absence or impairment of one or more of the primary perceptions constitutes color blindness; the characteristic of the defect depending upon which element is missing or impaired. The condition is congenital, does not disturb vision, is not dependent upon any demonstrable pathological lesion, is irremediable and is often hereditary. There are other forms of color blindness depending upon diseases of the retina and optic nerve which will be described in connection with those diseases.

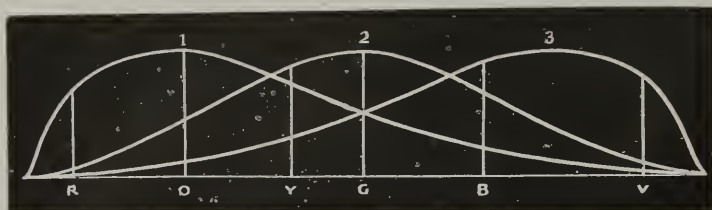


FIG. 29.—A diagram of color perception. 1, Red; 2, Green; 3, Violet; The height of the curve from the base line indicates the proportions in which the primary colors are mixed to produce the simple colors of the spectrum, red, orange, yellow, green, blue and violet.

CHAPTER III.

STRABISMUS.

Normally both eyes fix the same object. The image of the object looked at falls upon the fovea centralis of both eyes. This is accomplished by the co-ordination and association of movement of the six external ocular muscles of each eye. In looking up, down, right or left, the eyes move together and binocular or single vision results, because the images of objects in the field of vision fall upon identical parts of each retina, the upper half of the right retina corresponding to the upper half of the left, the right or temporal side of the right retina to the right or nasal side of the left, etc. If this association of movement is disturbed, so that the image of an object falls upon the fovea centralis of one eye and not upon the fovea of the other, we have strabismus or squint; and the vision of both eyes being good, diplopia or double vision may result. The eye which receives the image of the object looked at upon its fovea is called the fixing eye, the other is called the squinting or deviating eye. The deviation may be in any direction, depending upon which muscle or set of muscles is affected.

Strabismus is either Paralytic or Concomitant.

PARALYTIC STRABISMUS

Is produced by loss of power in one or more of the ocular muscles. Movement of the eye in the direction of the action of the affected muscle is limited or lost. The normal association of movement of the two eyes is disturbed and diplopia established. The confusion of the true and false image produces vertigo, and a peculiar carriage of the head is resorted to to favor the afflicted muscle and thus over-

come the diplopia. Generally the limitation of movement in a given direction will reveal which muscle is paralyzed, but sometimes the only guide is the position of the false image, which is always in the direction of the action of the crippled muscle.

The cause of Paralytic Strabismus is a lesion of the third, fourth or sixth nerve, and may be in the brain or along the track of the nerve. It is due to syphilis in about half of all cases and in the other half to tumors, rheumatism, diphtheria, poisons, disease of the spinal cord and injuries. Treatment will depend upon the cause, but the most useful remedies are iodide of potassium and strychnine.

CONCOMITANT STRABISMUS

Is due to the absence of normal association of movement of the ocular muscles without loss of their power. The muscles have their normal strength, but they do not work together so that each eye will fix the same object at the same time. The deviation may be monolateral or alternating, if the latter, vision in each eye will probably be the same. It is a condition which usually manifests itself early in life, the average age being about three years. Diplopia is rare in concomitant strabismus; the squinting eye is often so amblyopic as not to perceive the false image, and if it has good vision, concomitant squint arises so early in life that there is developed, with the growth of the child, a power of the mind to exclude the false image. There is more to discover relative to the cause of concomitant strabismus than has yet been revealed but the following may be given as etiological factors:

1. In three-fourths of all cases of internal strabismus there is hyperopia. Convergence and accommodation are associated actions, and in hyperopia there is excessive accommodation and the associated convergence sometimes amounts to internal strabismus. Relief of the hyperopia by convex glasses will at times straighten these eyes. But

that hyperopia is not a prime factor in the production of strabismus is proven by the fact that, in the majority of cases, the correction of the hyperopia by glasses has no effect on the strabismus, and also that there are so many cases of high degree of hyperopia in which there is no strabismus.

2. We find myopia associated with divergent strabismus and the explanation is that the convergence necessary to focus at the far point of a myopic eye, which is very close to the face, puts too great a strain on the internal rectus muscles and one of them gives up and divergence results. The exception to this rule is proven by the fact that only a very small proportion of the cases of myopia diverge.

3. In many cases of strabismus (72 per cent, Nagel) there is poor vision, or amblyopia in the squinting eye and the weight of authority is in favor of the view that the amblyopia is congenital and is the cause of the strabismus, the stimulation to binocular vision not being present. However a respectable minority claim the amblyopia to be the effect of non use of the squinting eye and not the cause of the strabismus. But that congenital amblyopia is only a factor in the production of squint is proven by the absence of squint in the majority of amblyopic eyes.

4. Another possible element in the production of concomitant strabismus is the unusual development of a single ocular muscle, the internal rectus being often unnaturally strong in internal strabismus and the same is true of the external rectus when the deviation is outward.

Treatment.—Rarely concomitant strabismus disappears without medical aid. Sometimes it is entirely removed by wearing the glass which corrects the refractive error, consequently these eyes should be tested and the proper glass ordered as soon as the child is old enough to wear them. The treatment for the remaining cases is operative, tenotomy of the over active muscle or advancement of the weaker one. Operations for strabismus should not be per-

formed before twelve or fifteen years of age, except in rare cases, owing to the tendency to development of over effect with the growth of the child.



FIG. 30.—Strabismus hook used to pick up the ocular muscle in performing tenotomy.

INSUFFICIENCY OF THE OCULAR MUSCLES.

This condition differs from strabismus in that the tendency to diviation is overcome by the desire for binocular vision. There is lack of balance of the ocular muscles but parallelism of the visual lines is maintained. The effort to maintain this parallelism and secure binocular vision results in asthenopia or painful vision, which may be manifested by pain over the insertion of the weak muscle, blurred vision, inability to do close or protracted work, photophobia, sub-acute congestion of the conjunctiva, headaches and vertigo and other neuroses. In late years much has been written about the relationship between insufficiency and chorea and epilepsy.

Treatment of this condition is difficult and belongs to the oculist. It consists mainly in constitutional improvement, exercise of the weaker muscle with prisms or partial tenotomy of the stronger.

NYSTAGMUS.

This condition is characterized by rapid, involuntary oscillation of the eye balls, generally in the lateral direction. It may be congenital or acquired and nearly always affects both eyes. If acquired the patient will, at first, complain of the movement of objects looked at.

Cause.—Defective development of the eyes, albinism, bad vision from corneal and lenticular opacities, blindness,

protracted use of the eyes in an abnormal position, it being common with miners, who work with their eyes directed obliquely upward. It is also due to brain lesions of central origin, ataxia and tumors of the cerebellum.

Treatment.—Improve vision by all possible means; if there is any refractive error put on the correcting glass, if there is a central corneal scar make a false pupil. In case of strabismus do a tenotomy, and if the occupation is at fault change it at once. The great majority of cases of nystagmus get little or no relief.

CHAPTER IV.

DISEASES OF THE LIDS.

BLEPHARITIS.

This is an inflammation of the lid border characterized by the following symptoms given in the order of their severity. 1. Hyperaemia. 2. Seborrhœa or yellow crusts on the border of the lid. 3. Ulceration at the root of the lashes. 4. Thickening of the edge of the lid. 5. Falling of eye lashes, with atrophy of their follicles. 6. Ectropium with eversion of the lachrymal puncta.



FIG. 31.—Blepharitis, eye lashes matted into bundles by the secretion along lid borders.

Cause.—It is sometimes eczematous in nature and is most frequently found in the fair skinned and strumous. The most prominent factor in its production is some error of refraction, usually astigmatism or hyperopia.

Treatment.—Correct the refractive error. Treat the constitution if due to struma. If there are ulcers around the hairs pull them out and touch ulcers with nitrate of silver stick. Rub into edge of lids, once or twice a day, an ointment of yellow oxide of mercury as follows:

R̄ Hydrargyri oxidi flavi,..... gr. viii
Vaselini,..... ʒ iv

Protect eyes from the irritation of dust, smoke, etc.

HORDEOLUM.

A sty is an acute inflammation of the connective tissue, or of a gland at the lid border. It is in appearance and symptoms a small boil.

Cause.—Error of refraction, general debility, possibly germ infection.

Treatment.—Hot applications to bring inflammation to a focus, then open. Correct constitutional condition if debility exists. Correct refractive error. Give sulphide of calcium, $\frac{1}{2}$ grain twice a day or dilute sulphuric acid ten drops after each meal.

CHALAZION.

This small tumor of the lid is due to a chronic inflammation of a meibomian gland. The mouth of the gland becomes closed and there is a consequent accumulation of its contents. The contents are mucoid or purulent, sometimes fibrous and solid.



FIG. 32.—Chalazion of upper lid.

Treatment.—The contents may escape and the tumor disappear spontaneously, but more frequently it is necessary to cut down upon it and if it is soft, curette it, if hard and fibrous dissect it out. The incision may be made through the skin or conjunctiva.



FIG. 33 —Clamp used in removing small tumors from the lids.

ECZEMA.

This condition is most frequently met with in children who have phlyctenular ophthalmia, and in adults with an irritating discharge from the eye. The symptoms and treatment are the same as of eczema in other parts. Oxide of zinc ointment, yellow oxide of mercury ointment and painting with strong solutions of nitrate of silver, 20 grains to the ounce, are efficacious.

PHITHEIRIASIS.

Crab lice may get into the eye lashes and give rise to excessive itching; the consequent rubbing and scratching of the lids sets up a mild inflammation which may be mistaken for blepharitis. The lice and their eggs may be seen on the cilia. Treatment is to rub the lid border and lashes thoroughly with mercurial ointment every night until the parasites are killed.

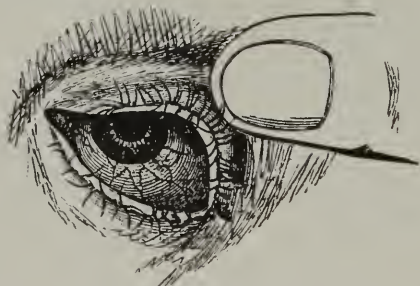


FIG. 34.—Trichiasis. (Wells.)

TRICHIASIS.

Wild hairs, misplaced or misdirected eyelashes rubbing the globe, produce great pain and blepharospasm and may cause ulceration and subsequent opacity of the cornea.

Trichiasis may be due to a chronic inflammation of the lid border of a proliferating type, which develops the false cilia as offshoots from the normal follicles. It may also be due to any cause which distorts the lid so that the lashes turn inward.

Treatment.—1. Epilation or pulling out of the offending hairs is only of temporary benefit, as they grow in again. 2. Electrolysis, introduced by Michel of St. Louis, is valuable. A needle attached to the negative pole is passed to the hair bulb, which is killed by a current of about five miliamperes. 3. Excision of the misplaced hairs with their bulbs is useful when they are few in number and close together. 4. If the wild hairs are isolated their direction can be changed by passing a needle, threaded with a loop, through the lid in the direction you wish the hair to take, then catching the hair in the loop and drawing it through the tissues as you pull the thread through. 5. If a distortion of the lid causes the lashes to offend, one of the operations for entropium should be done.

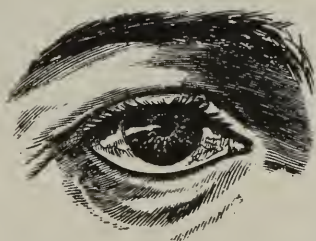


FIG. 35.—Entropium of the lower lid. (After Mackenzie.)

ENTROPIUM AND ECTROPIUM.

Entropium is a turning in of the lid. Ectropium is a turning out of the lid.

1. We have spasmodic entropium and spasmodic ectropium.

Spasmodic contraction of the fibres of the orbicularis near the lid border in conjunction with relaxed and flabby

skin and a deep set eye ball cause the lid border to turn inward, producing spasmodic entropium, which is nearly always found in the aged.

Spasmodic contraction of the fibres of the orbicularis farthest from the lid border, in conjunction with tense skin and prominent eye ball, cause the lid border to turn outward producing spasmodic ectropium, which is nearly always found in children and young people.

2. We have cicatricial entropium and cicatricial ectropium.

Trachoma, some forms of conjunctivitis and wounds may produce cicatricial contraction of the conjunctiva lining the lid which turns the edge of the lid inward, causing entropium.

Burns, wounds, ulcers, caries of the orbital border and other causes may produce cicatricial contraction of the skin of the lid, which turns the edge of the lid outward, causing ectropium.

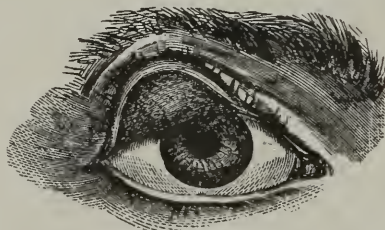


FIG. 36.—Ectropium of upper lid. (After Lawson.)

We have, in addition to the above conditions, a paralytic ectropium due to paralysis of the orbicularis muscle. The lower lid falls outward and away from the globe by its own weight. The lower lid is the only one affected by paralytic ectropium.

The treatment is almost always operative.

BLEPHAROSPASM.

Spasm of the orbicularis may be tonic or clonic. Tonic spasm is a reflex action, due to the irritation of corneal and

conjunctival diseases, foreign bodies, errors of refraction and to any condition which can cause photophobia. Treatment is to remove the cause.

Clonic spasm is paroxysmal and very painful. At given intervals the cramp seizes the orbicularis and other muscles of one side of the face causing distortion and great pain. The paroxysm passes off in about a minute to be repeated again after an interval varying in length in different cases. I have seen it return four or six times in an hour. The cause is some nerve or brain lesion and treatment is very ineffectual. Iodide and bromide of potassium have each been beneficial. If any peripheral exciting cause can be discovered it should be removed. Clonic spasm of the orbicularis belongs more properly to the realm of the neurologist.

LAGOPHTHALMIA.

Paralysis of the orbicularis, by constant exposure of the globe causes conjunctivitis, ulceration of the cornea, and an overflow of tears, due to the mal-position of the puncta. The cause is a lesion of the seventh nerve. The course of this nerve is long and devious and it passes through numerous tissues, which exposes it to accident or disease. The treatment consists in removing the cause meanwhile protecting the cornea from irritation by covering the eye with a bandage or holding the lids together with adhesive plaster.

PTOSIS.

This is a drooping of the upper lid due to a partial or complete paralysis of the levator palpebræ superioris muscle. This muscle is supplied by the third nerve, hence the condition is due to involvement of that nerve, usually from syphilis. There is also a congenital ptosis due to deficient development or absence of the levator palpebræ superioris.

Treatment.—Anti-rheumatic and anti-syphilitic. Electricity, one pole back of the ear and the other over lid, using a weak current. If these fail resort to an operation.

SYMBLEPHARON.

This is a cicatricial adhesion between the conjunctiva of the lid and the conjunctiva of the ball and is the result of the apposition of two raw surfaces, which may have been produced by operations, ulcers, burns, etc.



FIG. 37.—Symblepharon (after Mackenzie.)

BURNS.

Burns of the conjunctiva are serious because they lead to the adhesion between the lids and globe just described.

Powder burns may only involve the outside of the lids and may, if the eye is not closed quickly enough, seriously damage the cornea and entail loss of sight. The burns of percussion caps and torpedoes are especially destructive, owing to the added evil of the chemical action of the fulminate of silver and mercury of which they are made. All the foreign particles should be carefully picked out of the skin and cornea, an anodyne given to control the pain, and the eye put up in an aseptic oil dressing. If the cornea is much injured atropine should be used, as there is danger of secondary iritis.

Lime burns must be washed copiously with tepid water and all particles picked out with forceps if an anæsthetic has to be given to accomplish it. An anodyne can be given and cocaine used locally. Adhesions should be broken every day and sweet oil dropped between the lids. If the burn is deep symblepharon will follow.

Burns by acids should be thoroughly cleansed with tepid water and the raw surfaces, pain and inflammation combated as in the case of lime burns. Atropine should always be used where there is danger of iritis.

ECCHYMOSIS OF THE LIDS.

A "black eye" is the result of any cause which ruptures a blood vessel of the sub-cutaneous tissue of the lid. It is most frequently due to a blow. Time is the only cure. A bandage, cold applications, arnica or lead and opium wash will assist.

Erysipelas, abscess, syphilitic sore, ulcer, nævus, epithelioma, sarcoma, lupus, herpes zoster and molluscum contagiosum are all diseases that may be found on the lids but their descriptions belong more properly to a work on skin diseases.

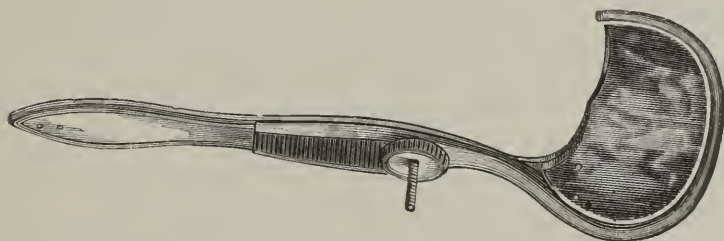


FIG. 38.—Clamp used to prevent hæmorrhage and steady the part in lid operations.

CHAPTER V.

DISEASES OF THE LACHRYMAL APPARATUS.

The lachrymal system is divided into a secretory part, the glands, and an excretory or drainage part, the puncta, canaliculi, sac and duct. Normally the lachrymal secretion is about balanced by evaporation. In acute diseases of the eye and under the influence of certain emotions there is pronounced hypersecretion of tears, or lachrymation, and the excretory apparatus being unable to carry them off they overflow on to the cheeks. In chronic eye diseases there is a slight hypersecretion of tears and if the drainage apparatus be normal they will be carried off through the natural channels. If the drainage is impaired, by eversion or occlusion of the puncta, plugging or stricture of the canaliculi or stricture of the duct, etc., the tears cannot find a natural outlet and the eye is bathed in tears which drip over the edge of the lid. This condition is called epiphora.

INFLAMMATION OF THE LACHRYMAL GLAND.

This occurs very rarely. There would be the usual symptoms of inflammation which might result in suppuration of the surrounding connective tissue or recover without it. There is often difficulty in excluding orbital cellulitis, phlegmon of the lid and periostitis. Treatment consists of hot bichloride fomentation, anodynes and evacuation of the pus if formed.

DISLOCATION OF THE LACHRYMAL GLAND.

If this occurs it appears as a tumor under the ocular conjunctiva at the upper and outer part of the globe. Treatment does not avail.

DACRYOCYSTITIS.

This is a catarrhal inflammation of the sac and duct. Stricture of the duct and lachrymal fistula will also be included under this head as all of these conditions merge into each other and are more or less inter-dependent. A slight catarrhal inflammation of the mucous membrane of the sac and duct, creates a muco-purulent discharge, some of which passes backward through the puncta and produces a mild conjunctivitis and epiphora. This slight attack may disappear without treatment. If the inflammation is more severe, the swelling of the mucous membrane will

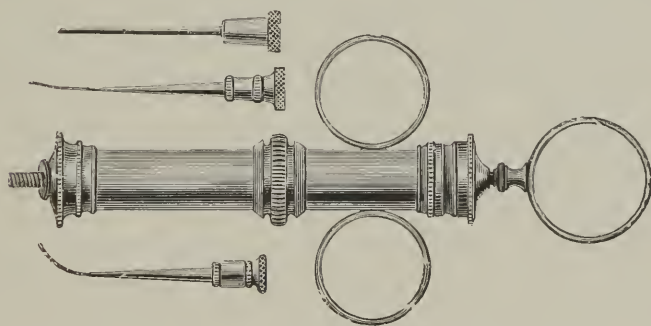


FIG. 39.—Anel syringe, used to wash out the lachrymal sac and duct.

cause a stricture of the duct and a consequent accumulation of the contents of the sac. The muco-purulent contents will become purulent, and will escape through the puncta and excite a conjunctivitis. With this conjunctivitis there will be hypersecretion of tears and epiphora. The accumulation of fluid in the sac produces a tumefaction which will disappear upon pressure, as the fluid is forced back through the puncta or through the stricture into the nose. If the tumor grows, the skin over it becomes thin and translucent and may break, emptying the contents of the sac and establishing a fistula. The purulent contents of the distended sac are extremely toxic and will almost surely infect a wound of the cornea, will often light up an act-

ive inflammation of the connective tissue surrounding the sac, and will, if of long standing, produce caries of adjacent bone.

Cause.—Dacryocystitis may be started by any of the numerous causes of inflammation of mucons membrane, such as temperature changes and infection. Stricture of the duct will of course set up dacryocystitis, and stricture may be due to morbid conditions of the nasal cavities, traumatism, asymetry of the face, deflected septum, periostitis or syphilis. The prognosis in chronic cases is bad. If cured they require months of treatment, and too often patients have not time or inclination to resort to the needed measures.

Treatment.—Teach patients to keep the sac empty by pressure. See that the nasal cavities are kept clean by washing them out with Dobell's solution. Drop into eye three times a day a 1 to 2000 solution of blue pyoktanin. Wash out sac with 1 to 5000 solution of bi-chloride of mercury every other day. If the discharge is purulent, inject into the duct a small quantity of a solution of nitrate of silver (gr. 10 to the ounce) after having cleaned it out by washing with bi-chloride of mercury or boracic acid solutions. If there be a stricture of the duct slit the canaliculus and dilate stricture with Bowman's probes.

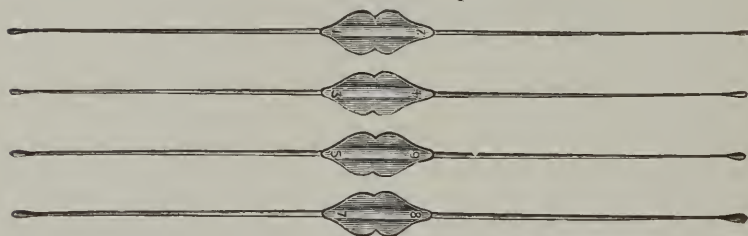


FIG. 40.—Bowman's probes for dilating the lachrymo-nasal duct.

CHAPTER VI.

DISEASES OF THE CONJUNCTIVA.

CATARRHAL CONJUNCTIVITIS.

This is the most frequent disease of the eye. It usually attacks both eyes, varies greatly in severity and duration, and tends to spontaneous recovery, rarely lasting over two weeks. Hyperaemia of the conjunctiva is generally given as a separate disease but practically differs from simple catarrhal conjunctivitis only in degree, being milder.

Symptoms.—A scratchy, burning feeling as of a foreign body in the eye. Congestion of the palpebral and ocular conjunctiva, the pericorneal zone being normal or the last part to become injected. Possibly some swelling of the lids. A muco-purulent discharge which mats the lashes into small bundles and sticks the lids together during sleep. Photophobia. Slight loss of vision due to the presence of mucous and pus on the cornea.

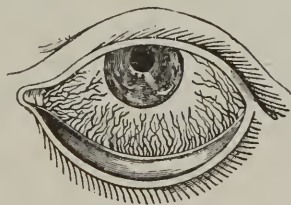


FIG. 41.—Conjunctival congestion. (After Guthrie.)

Cause.—Foul atmosphere, dust, smoke, wind, heat, cold, the glare of the sun and errors of refraction. The exanthematous fevers, diseases of the lachrymal sac and duct, nasal catarrh and hay fever. Germ infection, the disease being readily conveyed from one person to another.

Treatment.—Remove cause if discovered. Rest eyes and keep them clean. Use a cold compress as follows:

R_y.

Acidi Borici,.....	ʒi
Tincturæ Opii deodoratæ,.....	ʒvi
Aquæ destellatæ, q. s. ft.....	ʒviii

This is to be applied to the outside of the closed lids on a thin cloth, folded once or twice, for fifteen minutes at a time, four times a day. The solution should be ice cold when used and the wet cloths changed every minute. Apply a weak yellow oxide of mercury ointment or boric acid salve to the edge of the lids at night to prevent adhesion. If discharge is profuse and purulent, paint everted lids, once a day, with a solution of nitrate of silver, five to ten grains to the ounce. Astringent collyria containing sulphate of zinc, tannin, alum, etc., are very popular. They are, in my estimation, not as useful as the above harmless application and are capable of mischief if, through an error of diagnosis, they are used in iritis, cyclitis or acute keratitis.

After the subsidence of the acute attack, a chronic catarrhal conjunctivitis may persist, the symptoms of which are the same as in the acute condition but less severe. The same line of treatment may be followed, but in addition to it, the palpebral conjunctiva should be touched, once a day, with a crystal of alum or painted with a weak solution of nitrate of silver.

PURULENT CONJUNCTIVITIS.

This condition may be divided into two forms. 1. The infantile variety or Ophthalmia Neonatorum, which arises between the third and eighth day after birth and generally attacks both eyes. 2. The adult variety or Gonorrhœal Ophthalmia, which may attack but one eye.

Symptoms.—Congestion and chemosis of the conjunctiva. Profuse discharge, first of a thin, ichorous character, then

of pure pus. Intense, hard swelling of the lids, at times so great that the eye can not be opened. Heat and pain. Possible ulceration of the cornea with the consequent scars and, if the ulcer perforates, intra-ocular infection and panophthalmitis may result.



FIG. 42.—Purulent conjunctivitis. (After Dalrymple.)

Cause.—Inoculation with gonorrhœal virus, the gonococcus of Neisser being found in the discharge. There are mild types which clinically simulate purulent conjunctivitis in which the gonococcus can not be demonstrated. If the gonococcus is absent in the infantile variety, the disease has been caused by a vaginal discharge other than gonorrhœal. Such cases are always mild. If the gonococcus is absent in the adult form, the disease must be due to other pus producing germs. It will sometimes follow mechanical and chemical accidents or badly treated catarrhal conjunctivitis.

Treatment.—Keep eyes clean by washing them out every half hour with boric acid solution, as follows:

R̄

Acidi Borici,..... ʒii

Aquae destillatae,..... ʒviii

or every hour with 1 to 5000 bi-chloride solution. Ap-

ply iced cloths continuously. If pain is very severe give adult an anodyne. If corneal ulcer appears and there is danger of perforation, use atropine if ulcer is central, and eserine if it is peripheral. If the swelling of the lids tends to shut off the circulation to the cornea, divide the outer canthus. During the period of pus discharge, if lids can be everted, paint them with nitrate of silver, ten grains to the ounce. After the acute symptoms have sub-

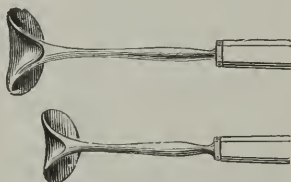


FIG. 43.—Desmarre's elevators used to raise the lids, for inspection of the eyeball, when they are thick and swollen, as in purulent conjunctivitis.

sided a condition akin to chronic catarrhal conjunctivitis will be found, which should be treated accordingly.

In adults, where one eye is affected, protect the good eye by covering it with a watch crystal held in position by adhesive plaster. To prevent ophthalmia neonatorum in a child born of a diseased mother, resort to the method of Crede, which is to wash its eyes thoroughly just after birth and drop between the lids, on the cornea, several drops of a ten grain to the ounce solution of nitrate of silver.

MEMBRANOUS CONJUNCTIVITIS.

The characteristic feature of this inflammation is a plastic, membranous deposit on the tarsal and sometimes on the ocular conjunctiva. It is customary to divide this affection into Croupous and Diphtheritic Conjunctivitis, but I think we are safe in considering them as the same clinically. In the diphtheritic type, the constitutional disturbance is profound, the symptoms more severe and the destruction of the tissue greater than in the croupous variety.

Symptoms.—Swelling of the lids; at times they are so

stiff and hard to evert that handling causes great pain. The discharge is at first ichorous and later purulent. The significant feature is a membranous deposit on the conjunctiva which may be in circumscribed spots or cover the whole conjunctival surface. The membrane may be rubbed off, leaving a bleeding surface, or may involve the whole depth of the mucous membrane and be impossible to remove. The cornea may ulcerate and the eye be lost by panophthalmitis. There may be considerable systemic reaction. When the membrane is finally thrown off, the disease simulates purulent conjunctivitis.

Cause.—Germ infection which may be epidemic or may rise from similar plastic inflammation of the throat and nose.

Treatment.—Clean eyes frequently with boric acid solution. Apply a solution of bi-chloride, 1 to 2000, to the everted lids three times a day, if it is possible to turn them. Use warm fomentations over the eyes. Sustain the constitution with tonics of iron, quinine and strychnine. When the membrane disappears, use nitrate of silver on the everted lids as in purulent conjunctivitis. If the disease is true diphtheria of the conjunctiva, a condition very rare in this country, institute a general diphtheritic treatment, anti-toxine, etc.

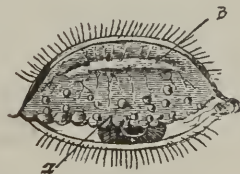


FIG. 44.—Granular upper lid a, granulations; b, line of scar, in typical position parallel with border of lid. (Nettleship.)

GRANULAR CONJUNCTIVITIS OR TRACHOMA.

The characteristic feature of this disease is hypertrophy of the conjunctiva and the appearance in that membrane of small granular bodies. These granules are composed of lymphoid cells and connective tissue cells surrounded by a

fibrous capsule. They are imbedded in the adenoid layer, and have a yellowish or grayish appearance. They develop later into connective tissue and together with the normal connective tissue layer, which has greatly increased in quantity, they undergo cicatricial contraction. This contraction distorts the tarsus and converts the normal membrane into a fibrous scar. Trachoma is generally slow and insidious in its development but may appear in an acute form.

Cause.—Contagion due to a micro-organism, most likely the micro-coccus discovered by Sattler.

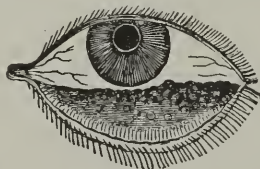


FIG. 45.—Granular lower lid. (After Eble.)

ACUTE TRACHOMA.

Symptoms.—Rapid swelling of the lids and hypertrophy of the conjunctiva. Pain, which may extend to the brow and temples, lachrymation, heat, photophobia and congestion, with a muco-purulent discharge. The palpebral conjunctiva is swollen, red and shiny. The translucent



FIG. 46.—Exuberant granulations. No indications of cicatrization are present. (Jones.)

granules, that are covered by the hypertrophied epithelium, usually are not seen until the acute symptoms subside, which occurs in from one to three weeks. It will often be impossible to distinguish this disease from acute catarrhal conjunctivitis until the granules appear.

Treatment.—Apply iced compresses. Cocaine locally is beneficial, but some cases reject it. Distress will sometimes be so great as to warrant the use of bromides or morphia. When the swelling and pain have subsided and the granules appear, treat as a case of chronic trachoma.

CHRONIC TRACHOMA.

Symptoms.—The eyes are irritable, giving distress under exposure or misuse. The lids are swollen and droop a little. There is a slight muco-purulent discharge and a scratchy feeling under the lids. Upon inspection of the palpebral conjunctiva, the trachoma bodies are found. In mild cases there will be few of these bodies scattered in

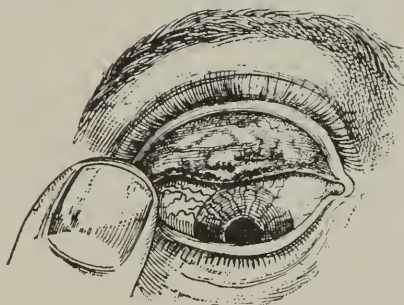


FIG. 47.—Trachoma and pannus. (Berry.)

the almost normal conjunctiva. In severer types they are larger and so abundant, especially in the retrotarsal fold, as to give the appearance of cauliflower. In the severest form there is inflammatory thickening of the palpebral conjunctiva. Its surface is rough and warty, due to the hypertrophied papillae and the trachoma granules. There is increase in the severity of all the subjective symptoms.

The ocular conjunctiva becomes injected and vision is much lowered by ulceration of the cornea and the development of pannus. Later, in the stage of cicatricial contraction, the conjunctiva becomes hard and fibrous and by its contraction bends the tarsus so as to produce trichiasis and its attendant evils.

Treatment.—As a germicide, use bi-chloride of mercury, 1 to 5000, dropped between the lids three times a day. To absorb the granules, which is a slow and tedious process, stimulate once a day by an application of nitrate of silver, ten grains to the ounce, or crystal of sulphate of copper. Ichthyol has lately been recommended, as follows:

R_x

Ichthyol,.....	℥iv
Glycerine,.....	℥iii
Aq. destillatae,.....	℥i

This should be applied to the everted palpebral conjunctiva once a day. Boroglyceride (30 to 50 per cent.) applied to the palpebral conjunctiva, once a day, is a useful remedy; as is also tannin and glycerine (30 grains to the ounce) used in the same way.

Where the appearance of the granules indicate its feasi-



FIG. 48.—Knapp's roller forceps.

bility, squeezing them out with Knapp's or Noyes' forceps facilitates the cure. This should be thoroughly done under an anaesthetic, and the lids subsequently treated with strong bi-chloride solution. Old cases in which there is considerable pannus, as shown in Fig. 46, are often greatly improved by the use of an infusion of jequirity. The cases on which jequirity is used should be carefully selected and as its use is sometimes attended with danger, this treatment should be left to an oculist. Some cases of trachoma seem incurable; they will relapse until the eyes are practically destroyed.

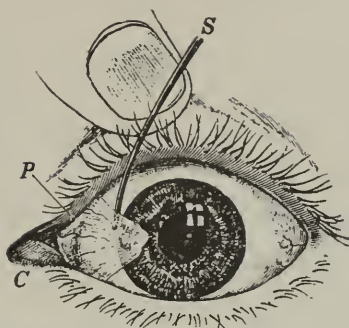


FIG. 49.—Pterygium. C, caruncle; P, puncta; S, probe passed under the upper margin. (Fuchs.)

PTERYGIUM.

This is a triangular mass of hypertrophied conjunctiva, the apex of which encroaches upon the cornea with the base generally in the inner, sometimes in the outer canthus. The head or apex is firmly attached to the cornea but a probe can be passed some distance under the upper and lower edge of the part over the sclera. It affects vision by growing in front of the pupil, or by traction produces astigmatism. If vision is not impaired and the growth is stationary, it need not be treated. If it is progressive, it should be removed. Laymen will always call this growth cataract. Our knowledge of the cause is, as yet, confined to theories. Treatment is surgical. For details of the numerous operations devised for this condition, consult a text book.

SUB-CONJUNCTIVAL ECCHYMOYSIS.

A hemorrhage under the conjunctiva may be due to strain, traumatism or disease of the blood vessels. It is seen often in children with whooping cough, and need cause no uneasiness. Coming on in an adult, without strain or accident, it indicates weakness of the vessel walls and portends hemorrhages in other organs, which might be of serious consequence. There is no pain attending the condition and treatment is unnecessary. Hot applications may hasten absorption of the clot.

PINGUECULA.

This is a small yellow elevation in the conjunctiva, generally found between the limbus of the cornea and the plica semilunaris, but is sometimes on the temporal side. It is composed of connective tissue and elastic fibres. It is of frequent occurrence, does no harm and need not be touched unless a patient insists upon its removal. This can be done with one snip of the scissors.

MORBID GROWTHS IN THE CONJUNCTIVA.

Under this head I will only mention the abnormal growths which may develop, as the diagnosis and treatment of these conditions lie along surgical lines. Syphilitic and tubercular lesions may be found in the conjunctiva, detection of the latter often requiring fine diagnostic discrimination. Epitheliomata and sarcomata may develop, and usually elect the limbus as their starting point. Thorough removal is imperative. Some cases demand sacrifice of the eyeball and orbital contents but even this does not always save the patient. Lipomata are found under the conjunctiva, between the superior and external rectus muscles and must be differentiated from a dislocated lachrymal gland. Papillomata may grow from any part of the conjunctiva, while Dermoid tumors are, as a rule, found as congenital formations, near the outer canthus. Cysts, Naevi and Angiomata are also found in the conjunctiva.

CHAPTER VII.

DISEASES OF THE CORNEA.

PHLYCTENULAR KERATITIS.

Clinically and etiologically, phlyctenular conjunctivitis and phlyctenular keratitis are the same disease, the only difference being the location of the vesicle. It is eczematous in nature and is found most frequently in strumous, delicate or poorly fed children.

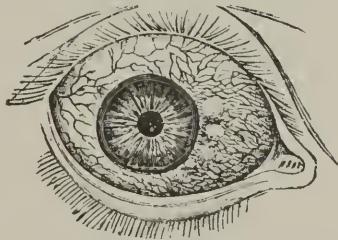


FIG. 50.—Phlyctenular conjunctivitis. (After Dalrymple.)

Symptoms.—The vesicle is at first a circumscribed accumulation of leucocytes, under the epithelial layer, but soon develops into an ulcer. The conjunctiva is injected, and there is a tendency of the enlarged vessels toward the phlyctene. Photophobia, pain and lachrymation are severe, which produces strong blepharospasm. Generally there is running of the nose, and eczematous scabs around the lips and nostrils. Usually the disease recovers in a few weeks without any injury to the cornea, but the result may not be so fortunate, and a deep ulcer may develop, followed by secondary iritis, perforation or staphyloma.

Cause.—It is a disease of childhood, and is supposed to be due to some irregularity of nutrition, the result of the strumous diathesis.

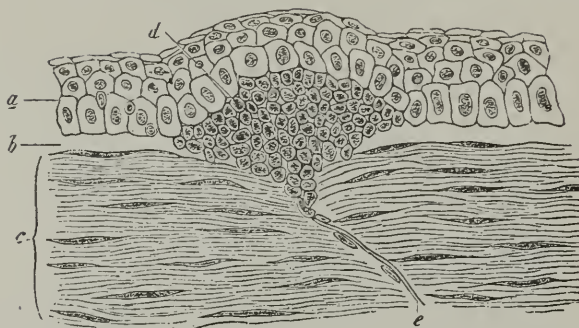


FIG. 51.—Phlyctenular keratitis. a, epithelial layer; b, membrane of Bowman; c, substantia propria; d, accumulation of leucocytes; e, corneal nerve. (Iwanoff.)

Treatment.—The cause being constitutional, give fresh air, wholesome food, tonics of syrup of the iodine of iron, malt or cod liver oil and keep bowels regular. Promote health in every way. Use locally hot fomentations and a weak ointment of yellow oxide of mercury rubbed in gently once a day.

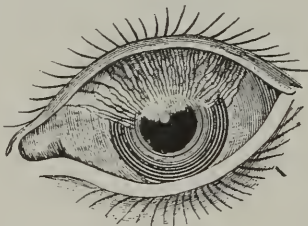


FIG. 52.—Pannus. (Fick.)

VASCULAR KERATITIS OR PANNUS.

The upper half of the cornea is the part most frequently affected, but its whole surface may be involved. It becomes grayish in color from cellular infiltration and covered by a mesh of fine blood vessels, which grow from the conjunctiva. The infiltration and vascularity are found between the epithelial and Bowman's layers, but may go deeper. If the new growth invades the substantia propria permanent scarring is the result. Vision is impaired and

may be reduced to light perception. As pannus is secondary to some other ocular disease the general symptoms will be those of the primary affection.

Cause.—Long-continued irritation of the cornea from trachoma, persistent phlyctenular keratitis, ingrowing lashes, exposure from imperfect closure of the lids, etc. The most frequent cause is trachoma.

Treatment.—Attend to the primary disease. The use of an infusion of jequirity produces a severe and dangerous purulent inflammation, which often results in great improvement and sometimes cure of the pannus, but this treatment should be left to an oculist.



FIG. 53.—Interstitial keratitis. (Nettleship.)

INTERSTITIAL KERATITIS.

It is a disease of childhood, but may be found in early adult life. The substantia propria is the part primarily involved. The duration of an attack may cover weeks or years.

Symptoms. — Circumcorneal hyperaemia. Pronounced loss of vision, due to haziness of the cornea, which may appear grayish from lymphoid infiltration, red from blood vessels in its tissue, or buff color from pus. Photophobia and pain are present, but are not commensurate with the gravity of the disease. Secondary iritis may set in and synechia occur.

In the syphilitic cases there will be the usual symptoms, such as glandular enlargement, sunken nose and ozaena, Hutchinson's teeth and scars at the angles of the mouth. The cornea is rarely restored, usually a scar more or less dense is left and staphyloma may result.



FIG. 54.—Hutchinson's teeth.

Cause.—Deranged nutrition dependent in the great majority of cases upon inherited syphilis. In the remaining cases upon scrofulous, tubercular or rheumatic constitutions and acquired syphilis.

Treatment.—Fomentations, as hot as can be borne, kept over eyes about half the time. Use just enough atropine to keep the pupils dilated. After all acute symptoms have subsided rub into eyes, once a day, an ointment of yellow oxide of mercury. Assume the existence of syphilis and give anti-syphilitic remedies, with tonics, good food and good air.

SUPPURATIVE KERATITIS, ULCERS OF THE CORNEA.

These two conditions can be described together.

Symptoms.—A yellowish spot, with hazy borders, appears on the cornea. There may be more than one ulcer. There is circumcorneal redness, pain, photophobia, lachrymation and dimness of vision. The loss of vision depends much upon the location of the infected area. Secondary iritis may set in. In some severe cases, where there is pronounced purulent infiltration, there may be pus in the anterior chamber. Pus in the anterior chamber is called hypopyum. The ulcer may perforate, and with the loss of

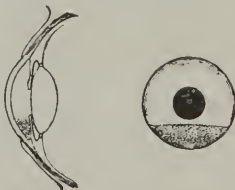


FIG. 55.—Hypopyon, seen from the front, and in section, to show that the pus is behind the cornea. (Nettleship.)

the aqueous humor the iris may be floated into the opening, and, if held there, we have an anterior synechia. With

perforation the lens may escape. In severest cases there is suppuration of the interior of the eye, which means its destruction and subsequent shrinkage.

Cause.—The primary cause is germ infection from purulent conjunctivitis, dacryocystitis, erysipelas, diphtheria, ozaena, pyaemia (by metastasis), septic fingers, handkerchiefs and instruments, or from an unknown source. The secondary cause may be a condition by which the cornea is rendered more susceptible to infection, such as debilitating diseases, marasmus, injuries from foreign bodies and operations, and anaesthesia from paralysis of the fifth nerve. The ulceration due to paralysis of the fifth is called neuroparalytic keratitis. With paralysis of the fifth there is loss of sensation and foreign bodies are no longer removed from the cornea, by the reflex action of the lids, and abrasion results. Abrasion is further facilitated by the dryness of the cornea which exists in the absence of winking. A condition, similar to paralysis of the fifth, in its results to the cornea, is found in lagophthalmia or paralysis of the seventh.

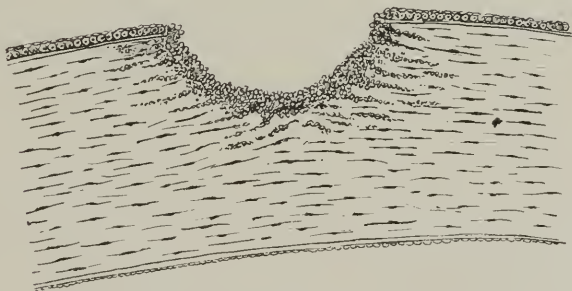


FIG. 56.—Ulcer of the cornea. The epithelium, Bowman's membrane and part of the substantia propria are gone. The floor of the ulcer is infiltrated with pus cells.

Treatment.—Hot fomentations. Judicious use of cocaine. Wash out conjunctival sac every three hours with a solution of bichloride of mercury, 1 to 5000. Use atropine to combat iritis and to draw the iris away from a cen-

tral ulcer, which threatens to perforate. Use eserine if the ulcer is peripheral. The focus of germs may be destroyed by touching the ulcer with the actual cautery, with silver nitrate, grains 30 to the ounce, with 20 per cent solution of carbolic acid, or by scraping it out with a small curette. Paracentesis is sometimes beneficial by relieving tension. Build up the constitution on iron, quinine, strychnine, etc. If the ulceration is neuroparalytic or due to paralysis of the seventh nerve, the cornea must be protected by the lid and a bandage or adhesive plaster will be necessary to accomplish it.

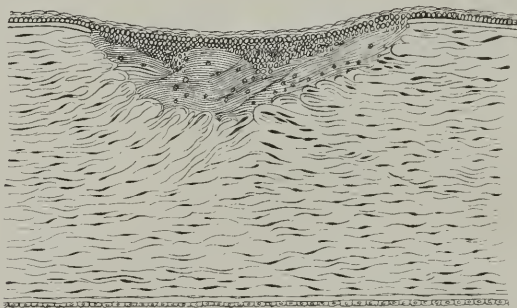


FIG. 57.—The cornea after ulceration, showing the scar tissue.

OPACITIES OF THE CORNEA.

Nebula, macula and leucoma are names given to opacities of the cornea. These opacities represent scar tissue, and are left after certain diseases and injuries. If the first two layers of the cornea are destroyed they may heal without leaving any sign, but any loss of the substantia propria is repaired with scar tissue. The amount of damage to sight produced by an opacity depends upon its location relative to the pupil. Recent scars are improved by time and direct massage with a stimulating ointment, but old

ones will remain unchanged. If the opacity is central and there is any peripheral clear cornea, an artificial pupil will restore vision.



FIG. 58.—Total staphyloma. (Fick.)

STAPHYLOMA.

Severe ulceration of the cornea so decreases its power of resistance that the normal intra-ocular pressure may cause it to bulge forward, destroying the natural curve. The distension may involve the whole cornea or only a part of it. When staphyloma is the result of a perforating ulcer, the iris may be caught in its tissue and severe pain and intra-ocular inflammation result. The staphylomatous cornea is never transparent. It may be stationary or progressive, small or so large that the lids will not close over it. In some cases nothing need be done. The treatment is operative.

ARCUS SENILIS.

A narrow white ring is often seen near the circumference of the cornea. It is usually found in old people but may occur in the young. It is caused by hyaline degeneration and requires no treatment. It has no influence on the healing of wounds, as for example the incision in cataract operation.

CONICAL CORNEA.

Sometimes the center of the cornea becomes weakened by an atrophic process and the intra-ocular pressure pushes it forward; the convex sphere changing to a cone. The

cornea remains clear except for the occasional appearance of a nebula at its apex. The process is slow and gradual but finally reaches a point where it stops. Vision is greatly impaired. Inspection of the eye reveals no abnormality, except in pronounced cases, when a side view will show its conical form. Diagnosis in the early stages is difficult and treatment not very effective. The latter should be left to an oculist.

FOREIGN BODIES IN THE CORNEA.

It is very common for cinders, sand, pieces of emory, iron, steel, etc., to become lodged in the cornea. Pain and lachrymation will be intense, with more or less circumcorneal injection. If simple inspection does not reveal the offender use oblique illumination. This is done by seating the patient about two feet from a light and with a 16 or 20 Dioptre convex lens, focus the rays *obliquely* on the part to be examined. In this way the cornea, iris and lens may be thoroughly inspected. To remove a foreign body, the cornea should first be anaesthetized by several drops of a solution, as follows:

R_x.

Cocaine hydrochloratis,..... gr. v

Aquae destillatae,..... ʒii

then, with a needle or spud, pick it out with as little destruction to corneal tissue as possible. When the epithelium is denuded there is always danger of infection, therefore an antiseptic wash (solution hydrarg. bichloride 1 to 5000) should be used for three or four days or until the epithelial layer is restored.

CHAPTER VIII.

DISEASES OF THE SCLERA, IRIS AND CILIARY BODY.

DISEASES OF THE SCLERA.

EPISCLERITIS.

Between the conjunctiva and the sclera is a loose connective tissue called the episclera. Inflammation may only attack this tissue or may go deeper, into the sclera proper.

Symptoms.—In episcleritis there appears a patch of dusky red injection under the conjunctiva, generally between the insertion of a rectus muscle and the cornea. The discharge is watery and pain and photophobia, if any, are slight. The inflamed spot may disappear spontaneously, may persist for weeks, has a tendency to recur, and will often leave a gray, discolored patch.

Cause.—Rheumatism, gout, scrofula and menstrual derangement. It may arise from exposure to the weather and is also said to appear over the insertion of a rectus muscle suffering from insufficiency. Frequently the cause is obscure.

Treatment.—Some cases are so mild as to need no treatment. The constitutional cause, if discoverable, should be attended to. Correct muscular anomalies and refractive errors. Apply hot fomentation. Use atropine if there is any tendency toward iritis. When chronic, stimulation with yellow oxide of mercury ointment is useful.

SCLERITIS.

Inflammation of the sclera may be circumscribed or diffuse.

Scleritis Circumscripta resembles episcleritis but the symptoms are all more severe and, as a deeper structure is inflamed, there is much greater danger of involvement of the

uvea. The discharge is watery and pain and photophobia may be pronounced. The inflammation may extend to the underlying uveal tract and produce iritis and cyclitis; or extend to the cornea, producing a haziness of its deep layers. The condition is usually chronic and the scleral wall may become thinned and staphyloma follow.

Scleritis Diffusa is a rare disease and is generally acute. The redness will cover the entire white of the eye. Care must be taken not to confound it with conjunctivitis or iritis. The symptoms are about the same as in the circumscribed form, there being the same tendency to involvement of the cornea and uveal tract and the same possibility of staphyloma.

Cause.—The cause of both forms is generally rheumatism, gout, syphilis or scrofula.

Treatment.—For the syphilitic form use mercury and iodide of potassium. In scrofulous cases, tonics, good air and good food. When due to rheumatism, salicylate of sodium, Rochelle salts, etc. If gouty in origin, iodide of potassium or colchicum. Combine above treatment with hot baths, warm fomentations over eyes, leeching of the temples, and locally, cocaine and atropine. Never put irritants, such as zinc or copper in these eyes.

DISEASES OF THE IRIS.

Mydriasis or dilatation of the pupil may be due to many causes among which are:

1. The use of drugs called mydriatics, such as atropine, homatropine, scopolamine and cocaine.
2. Increase of intra-ocular pressure as in glaucoma.
3. Loss of vision as in atrophy of the optic nerve.
4. Paralysis of the third nerve.
5. Dimness of light.
6. Ingestion of certain drugs, belladonna, ergot, etc.
7. Apoplexia in the later stages.

Myosis or contraction of the pupil may be due to:

1. The use of drugs called myotics, such as eserine and pilocarpine.
2. Evacuation of the aqueous humor.
3. Hyperaemia of the iris as in iritis.
4. Paralysis of the cervical sympathetic nerve.
5. Bright light, accommodation and convergence.
6. Ingestion of certain drugs, as opium and alcohol.
7. Apoplexia in the early stages.

The Argyll-Robertson pupil is one which responds to convergence but not to light, and is significant of locomotor ataxia. The variations of mydriasis and myosis dependent upon irritation and disease of the brain and spinal cord are too complex to dwell upon here.

Anterior synechia is an adhesion of the iris to the cornea, due to perforation of the cornea and lodgment of the iris in the wound.

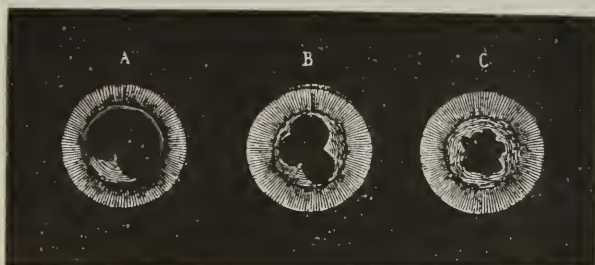


FIG. 59. Various forms of posterior synechia. A, single attachment; B, Multiple attachment forming the so-called "rose-water" pupil; C, irregular annular attachments [Sichel.]

Posterior synechia is an adhesion of the iris to the anterior capsule of the lens.

In complete posterior synechia we have what is called exclusion of the pupil. Where the pupillary area is filled by a membrane, we have occlusion of the pupil. (Fig. 60)

IRITIS.

The disease may be divided by its course into acute or chronic; pathologically it may be plastic, suppurative or

serous; etiologically it may be divided into as many forms as there are causes, the leading varieties being syphilitic, rheumatic, gouty, idiopathic, traumatic and secondary. The typical form of iritis, is plastic; serous iritis, according to Collins, Priestley Smith and others, being more appropriately a cyclitis.

PLASTIC IRITIS.

Symptoms.—Injection of the deep blood vessels around the cornea, later extending over the entire white of the eye. Discharge of a watery character. Intolerance of light and pain of a neuralgic nature, beginning in the eye-ball and extending over the brow, temples and cheek. The pupil becomes small and will not react to light. Its normal color changes to a darker tone, a blue or gray iris becoming green. The aqueous becomes turbid from lymphoid cells, pus and red blood corpuscles, and vision is correspondingly impaired. Adhesion will take place between the iris and anterior lens capsule, constituting posterior synechia. If these adhesions are broken, pigment deposits will be left on the capsule of the lens. When the attack is syphilitic in origin, gummata may develop on the iris. If there is much pus in the aqueous humor it may settle in the anterior chamber, producing hypopium.

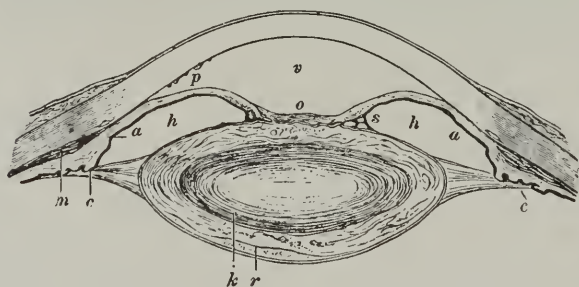


FIG. 60.—Exclusion and occlusion of the pupil, with bulging of the iris forward from accumulation of fluid in the posterior chamber. The posterior chamber (h) is thus made deeper, the anterior chamber (v) shallower, especially where the root of the iris (a) is pressed against the cornea. The pupil is closed by an exudate membrane o. (Fuchs.)

Sometimes the exudation in the anterior chamber leaves a membrane across the pupil which may be mistaken for cataract. Such a condition is spoken of as occlusion of the pupil. (Fig. 60.) Iritis may attack one eye or both. Its duration depends largely upon the treatment, but will generally last from two to six weeks.

Cause.—In fifty per cent of all cases it is due to syphilis, secondary, tertiary, acquired or inherited. The next most potent factors are rheumatism and gout.

It may arise as secondary to other eye diseases or be due to direct lesion, accidental or operative. Gonorrhoea and diabetes are also said to be etiological factors.

Treatment.—Prohibit work and protect eyes with shaded glasses. Look to the general health of patient, paying special attention to the condition of the alimentary canal. To prevent posterior synechia, dilate pupil with atropine and keep it dilated through the whole attack.

Leeching at the temple is sometimes efficacious. For the pain, give antipyrine or morphia and apply heat, dry or in the form of watery fomentation. In syphilitic cases give mercury and iodide of potassium. When rheumatic or gouty in origin, use the salicylates, colchicum, lithia, etc., combined with hot baths or pilocarpine sweats.

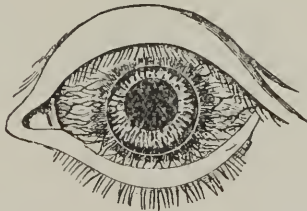


FIG. 61.—Circumcorneal injection, the deep blood vessels around the limbus being the ones involved. (After Dalrymple.)

SUPPURATIVE IRITIS.

This form is generally due to wounds or operations and does not differ materially from the plastic form, except that

the presence of pus infection makes the symptoms more severe and the prognosis very grave.

SEROUS IRITIS.

This disease and serous cyclitis are the same; not only are the iris, ciliary body and the choroid involved but also Descemet's membrane of the cornea. It has been described under the names Descemitis, Keratitis Postica, Keratitis Punctata, Serous Uveitis and Serous Irido-cyclitis.

Symptoms.—Slight pericorneal injection, pain insignificant, vision sometimes only a little below normal, increase of aqueous evidenced by unusual depth of the anterior chamber and plus tension. The pupil will not be contracted as in plastic iritis and the iris will only be slightly discolored. Posterior synechia may occur but is not as common as in other forms of iritis. There will also be found a characteristic deposit of pigment in the form of fine dots on the lower half of the posterior surface of the cornea, which constitutes Keratitis Punctata. The course of the disease is more or less chronic and the subjective symptoms mild as compared with the other forms of iritis.

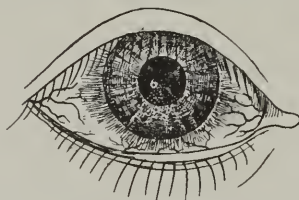


FIG. 62.—Serous iritis, showing ciliary injection and keratitis punctata.

Cause.—The causes are the same as in plastic iritis.

Treatment.—The same as in plastic iritis except that atropine must be carefully used owing to the danger of increasing the plus tension. If the tension becomes dangerous it may be reduced by cocaine locally, or pilocarpine injections to produce diaphoresis.

DISEASES OF THE CILIARY BODY.

Inflammation of the ciliary body is not an isolated condition but is probably always associated with disease of the iris or choroid. Cyclitis may be acute or chronic; plastic, suppurative or serous.

PLASTIC AND SUPPURATIVE CYCLITIS.

The symptoms of these two conditions are the same as in iritis, with the addition of opacity of the vitreous, severe pain upon pressure over the region of the ciliary body and characteristic tension, which is plus in the acute stage but later becomes decidedly minus, due to atrophy of the ciliary body and shrinkage of the vitreous. The lens sometimes becomes opaque and detachment of the retina may occur. Plastic cyclitis is dangerous, suppurative cyclitis is almost always fatal to vision. Treatment is the same as in iritis.

SEROUS CYCLITIS.

This is the same as serous iritis (see page 74).

SYMPATHETIC OPHTHALMIA.

Sympathetic Ophthalmia is a diseased condition arising in one eye, caused by some organic lesion of its fellow. The eye which is first affected is called the *exciting* eye, while the other is called the *sympathizing* eye.

The disease takes two forms—sympathetic irritation and sympathetic inflammation.

Symptoms.—

(1). Sympathetic irritation is a functional derangement characterized by intolerance of light, lachrymation and fatigue of the eye when used. Visual acuity may be impaired and sometimes temporary obscuration of sight occurs. There may be considerable pain, of a neuralgic character, in and around the eye and also some peri-corneal injection.

(2). Sympathetic inflammation is sometimes very slow and insidious in its development. When established there is intense circumcorneal injection, an inflamed iris,

opacities in the vitreous, intense neuralgic pain in the region supplied by the fifth nerve; also pain upon pressure over the ciliary region. As the disease progresses optic neuritis, choroiditis, synchysis of the vitreous and detachment of the retina develop.

Cause.—The cause is an inflammation of the uveal tract of the exciting eye. This uveitis may be idiopathic, but the inflammation most prone to excite sympathetic trouble is that due to a wound of the ciliary region or the presence of a foreign body in the exciting eye. Other sources of the exciting uveitis are perforating corneal ulcers and intra-ocular tumors. After an enucleation the optic nerve or ciliary nerves being caught in the cicatrix have been known to give rise to sympathetic irritation. Sympathetic ophthalmia may arise at any time from two weeks to many years after the lesion of the exciting eye. In spite of many theories our knowledge of how this inflammation is conveyed from one eye to the other is yet speculative.

Treatment.—As sympathetic irritation is always cured by enucleating the exciting eye, this should be done at once, but if sympathetic inflammation is established this procedure will rarely stop it and should not be resorted to if the exciting eye has useful vision, as it will often retain the best vision of the two. If, in sympathetic inflammation, the exciting eye is blind, enucleate it. Its removal may do some good and can do no harm. Patient should be kept in a dark room, hot fomentations used from four to eight hours a day, anodynes given for pain and mercury and tonics given internally. As the treatment of sympathetic inflammation is so unsatisfactory, its prophylaxis becomes doubly important and I would advise the enucleation of all blind eyes affected with chronic irido-cyclitis; all eyes with irido-cyclitis due to the presence of a foreign body, which cannot be removed, even if fairly good vision remains; also all shrunken globes and stumps which are tender on pressure.

CHAPTER IX.

DISEASES OF THE CRYSTALLINE LENS AND GLAUCOMA.

DISEASES OF THE CRYSTALLINE LENS.

DISLOCATION OF THE LENS.

This condition may exist congenitally or may be due to accident or disease. The lens may be partially held by the suspensory ligament or may be totally detached. The dislocation may be to the side, back into the vitreous or forward, through the pupil, into the anterior chamber. If due to disease it is associated with choroiditis, cyclitis and a fluid state of the vitreous. The lens may be perfectly transparent or cataractous. If cataractous the malposition may be easily detected. If transparent and not in the anterior chamber the ophthalmoscope will reveal the condition. A transparent lens in the anterior chamber can be diagnosed by close inspection with the unaided eye. Congenital partial dislocation may be left alone. When due to accident or disease it would better be removed.

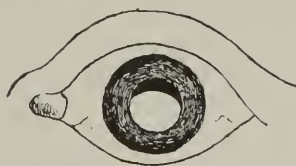


FIG. 63.—Downward dislocation of a cataractous lens. (Alt.)

CATARACT.

Opacity of the lens, or its capsule, or both, constitutes cataract. Numerous terms, which explain themselves, are used in classifying cataracts, such as lenticular, capsular and capsulo-lenticular; partial and complete; traumatic and spontaneous; fluid, soft and hard; congenital, juvenile and senile; immature, mature and hypermature; simple

and complicated; stationary and progressive; gray, white, amber and black.

Cataracts are further classified by terms which indicate the location of the opacity as follows;

Anterior Polar Cataract, in which the opacity is a small spot in the center of the anterior capsule and extending into the subjacent lens substance.

Posterior Polar Cataract, similar to the preceding, except located in the center of the posterior capsule.

Lamellar or Zonular Cataract, in which the opacity is confined to one of the layers of the lens.

Nuclear Cataract in which the opacity begins in the hard center of the lens.

Cortical Cataract, in which the opacity begins at the circumference of the lens.

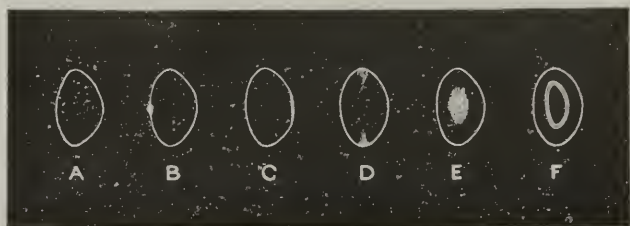


FIG. 64.—A, normal lens; B, anterior polar cataract; C, posterior polar cataract; D, cortical cataract; E, nuclear cataract; F, Lamellar cataract. (Juler.)

Symptoms.—In children, if the cataract is complete, it is easily diagnosed, as the pupillary area will be white or gray and the eye will be blind. If the cataract is zonular, which is the most frequent form in children, and is always congenital or arises in early infant life, the vision is much reduced, the child behaving as if near-sighted. Best vision is secured when the pupil is dilated, as the patient can then see around the opacity. Hence these children will shade their eyes or turn their backs to the light to get better vision, and will find their sight improved by the twilight or cloudy weather. Close inspection, with the pupil dilated,

will reveal a pale, round, central opacity of the lens. Anterior Polar Cataract is easily detected by the small, snow-white speck seen in the pupillary area. It does not affect vision as much as the zonular variety. Posterior Polar Cataract is generally secondary to some other diseased condition in the eye and vision is poor. It can not be seen except by oblique illumination or by the use of the ophthalmoscope. The great majority of cataracts arise after the 45th year and are called senile. They are usually nuclear or cortical. The first symptom noticed is failing vision, not improved by glasses. If nuclear, vision is improved by any circumstance which dilates the pupil. There is generally some hyperæmia of the conjunctiva, lachrymation and itching. Sometimes inspection of the pupil reveals no anomaly, but ordinarily cataract, in the advanced stage, shows a distinct white or grayish appearance of the pupillary area. Oblique focal illumination will demonstrate most opacities, but to determine their presence in doubtful cases the ophthalmoscope must be used. With the pupil dilated the whole lens can be brought under inspection and the slightest opacity will be detected by the ophthalmoscope. The time between incipency and maturity varies greatly in different cases.

Cause.—Cataract is supposed to be due to some disturbance of the nutrition of the lens. It is a degenerative change coming as do gray hairs, very little being known of the conditions that conduce to it. Diabetes, ergotism, heredity, glaucoma, hyperopia, and spasms in children are supposed to be etiological factors. Traumatic Cataracts are due to some accident which punctures the capsule or loosens the lens from its ligamentous attachment.

Treatment.—Spontaneous absorption of the cataract has been reported in a few authenticated cases, but no therapeutic agent has yet been discovered that will bring about this happy result. The treatment is surgical. For the operations suited to the different varieties consult a text

book. Before operating or recommending a case for operation be sure to test the bad eye for other pathological conditions. Cataract prevents the distinction of objects, but does not obstruct light. Take the patient into a darkened room and with the good eye well covered see if he can point out the direction of a lighted candle when held in all parts of the field of vision. If he can readily follow the light his retina and optic nerve are healthy and a successful operation will make him see again. If he can not see the light at all an operation is useless. If he sees only in certain parts of the field or detects slowly the difference between light and shadow an operation will be proportionately meager of results.

GLAUCOMA.

The normal resistance of the globe, when palpated by the index finger of each hand, is designated by the letter T. If the globe is abnormally hard its increased tension is designated relatively by the signs $T+?$, $T+1$, $T+2$, and $T+3$. If abnormally soft, by $T-?$, $T-1$, $T-2$, and $T-3$.

The tension of an eye depends upon intra-ocular pressure. Glaucoma is a diseased condition, due to excessive intra-ocular pressure.

About seventy per cent of the cases of glaucoma occur after the age of 50, and about the same per cent are in hyperopic eyes.

Glaucoma may be idiopathic or secondary to some other pathological condition of the eye. It may also be acute or chronic.

Symptoms.—In an acute case the attack generally begins at night with severe pain in the ball, radiating to the distribution of the fifth nerve. Vision is lowered, the field diminished and lights have rings of color or halos around them. The conjunctiva is injected, and may be chemotic. The cornea is hazy and not normally sensitive to touch. If the pupil can be seen it will be found dilated. The iris reacts slowly or not at all and has lost its brilliancy of color.

The anterior chamber is shallow, and tension of the globe decidedly increased. There may be constitutional symptoms, such as rise of temperature and pulse, and vomiting. In these cases the prognosis is very grave as vision may be completely destroyed in a few hours.

In simple or chronic glaucoma, which is the most frequent form, we have an insidious and progressive condition. At first the patient will notice an attack characterized by slight pain in the eye and forehead, with some congestion of the conjunctiva, dimness of vision and perhaps halos or colored rings around the lamp or gas flame. These symptoms will soon pass off to be followed again after a period varying from days to months by a similar attack most likely a little more severe. These attacks become more and more frequent and in the intervals the eye tends to return less and less to the normal, and thus by a simple continuance of the process, which may cover a period of from five to fifteen years, the full development of glaucoma is reached. When seen, if the disease is established, tension will be found plus, the pupil dilated and iris immobile, the reflex from the pupillary space greenish in hue, accommodation will be weak, the patient requiring stronger glasses for reading than the age demands, the field of vision will be contracted, especially on the nasal side, and with the ophthalmoscope we shall find the retinal veins large, and the arteries small and the characteristic cupping of the optic disc. (Figs. 65 and 66.)



FIG. 65.—Section of very deep glaucoma cup. (Nettleship.)



FIG. 66.—Ophthalmoscopic appearance of deep cupping of the disc in glaucoma. (Nettleship.)

Absolute glaucoma is the name used for the last state, in which the globe is stony hard, the lens possibly cataractous, vision lost even for light, and pain constant and distressing. An attack of acute glaucoma may occur during the course of simple chronic glaucoma or may arise without any premonitory symptoms.

Increased intra-ocular pressure due to some other disease of the eye is called secondary glaucoma. The prognosis in all forms of glaucoma is bad.

Cause.—The intra-ocular fluid is supposed to be secreted mostly by the vascular portion of the ciliary body, and is excreted chiefly through the spaces of Fontana, at the angle of the anterior chamber, into the canal of Schlemm. Normally the angle of the anterior chamber is as seen in fig-

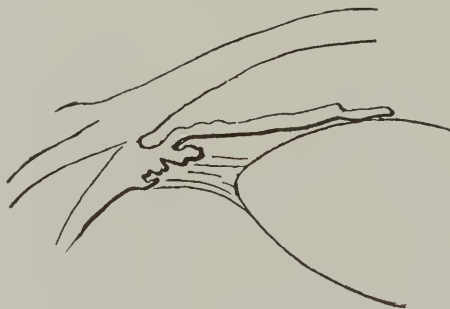


FIG. 67.—Angle of the anterior chamber in the normal eye. (Birnbacher.)

ure 67 but excretion through the spaces of Fontana may be cutoff by adhesion of the iris to the periphery of the cornea as in figure 68.



FIG. 68.—Angle of the anterior chamber in glaucoma closed by adhesion of iris-base to the periphery of the cornea. (Birnbacher.)

Hypersecretion, sub-normal excretion, or both, may produce increased intra-ocular pressure. Theories of the etiology of glaucoma may be divided into hypersecretion theories, and retention theories. The masters in ophthalmology have been divided in this war of hypersecretion versus retention. At present it is safest to say that some cases are due to one cause and some to the other and yet some to a combination of both. Secondary glaucoma may be directly attributable to intra-ocular hemorrhage, complete anterior or posterior synechia, traumatic cataract with rapid swelling of the lens, intra-ocular tumors, etc.

Treatment.—As soon as positive of the diagnosis, do a broad iridectomy. This procedure is the most curative measure at our disposal. Though introduced in 1856 by Von Graefe, and practiced ever since that time, we do not yet know exactly how it produces the amelioration which follows in the majority of cases. Next in value to the iridectomy is the use of sulphate or salicylate of eserine, grain one to the ounce. In chronic cases use one drop a day; in the acute form one drop every two or three hours. For pain use hot fomentations and anodynes.

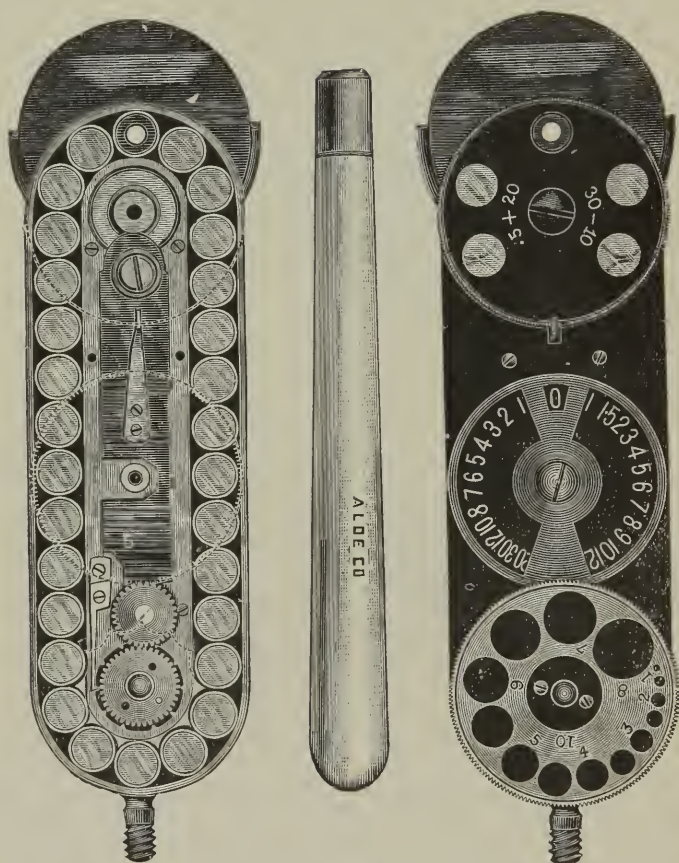


FIG. 69.—An ophthalmoscope (Morton's) used to view the interior of the eye.



Ophthalmoscopic Appearance of the Normal Fundus.

CHAPTER X.

DISEASES OF THE CHOROID AND RETINA.

DISEASES OF THE CHOROID.

The choroid is composed principally of blood vessels and pigment, and lies between the sclera and the retina. Its function is to nourish the retina and vitreous, and to prevent reflection by the power to absorb light possessed by its pigment. In Albinos there is almost total absence of pigment in the uveal tract, and great distress from photophobia is the result. Albinos are, as a rule, afflicted with amblyopia, refractive errors or nystagmus. Dark glasses are almost a necessity to these patients.

CHOROIDITIS.

If the morbid process is limited to the choroid, external signs of inflammation are absent, visual disturbance will be the only subjective symptom, and the objective symptoms will be revealed by the ophthalmoscope. The visual disturbance will consist of floating bodies in the field, or of one or more areas, in which vision is reduced or lost, called scotomata. There may also be distortion of the outline of objects called metamorphopsia.

The ophthalmoscope will show, in recent cases, ill-defined yellowish patches under the retinal vessels. These spots of exudation may absorb and leave no sign, but generally the choroid at these points atrophies and the sclera shows, glistening white, through it.

Around the borders of these atrophic areas, pigment is profusely deposited. The retina over the inflamed area is usually involved and also partakes of the subsequent atrophy, which explains the scotomata or blind spots in the field of vision.

The inflammatory process may extend to the vitreous and cause a cloudiness of that body, at times so dense as to prevent an ophthalmoscopic view of the underlying tissues.

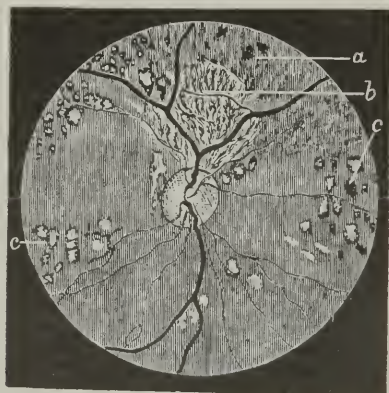


FIG. 70.—Atrophy after syphilitic choroiditis, showing various degrees of wasting. (Hutchinson). a, atrophy of pigment epithelium; b, atrophy of epithelium and chorio-capillaris; the large vessels exposed; c, spots of complete atrophy, many with pigment accumulation.

Cause.—Syphilis, malnutrition, scrofula, anaemia and high myopia, and in some cases no cause can be assigned with any degree of accuracy.

Treatment.—Absolute rest of eyes, tinted glasses, mercury, iodide of potassium and tonics.

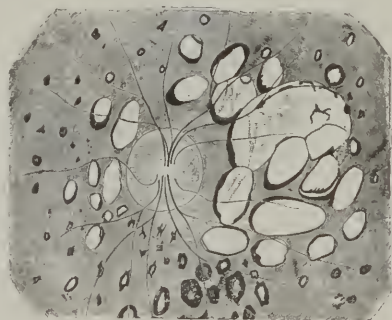


FIG. 71.—Atrophic patches in the choroid, (choroiditis disseminata.) (After Foerster.)

PANOPHTHALMITIS.

This condition is also called suppurative choroiditis, but, as the whole uveal tract, as well as all the other tunics of the eye are involved, I prefer the name used. It is an acute purulent inflammation of the contents of the globe in which vision is always lost.

Symptoms.—Intense pain, swelling of the lids and chemosis of the conjunctiva, with early loss of vision. If the process begins in an ulcer or wound of the cornea this tissue will soon become opaque and suppurative. If the lesion starts from within, the iris will change color as in iritis, and the aqueous become muddy, but in spite of this the yellow reflex caused by pus behind the lens may generally be seen.

Cause.—Intra-ocular pus infection from wounds, operations, perforating ulcers of the cornea or from pyaemia by metastasis.

Treatment.—Control the pain by leeches, hot bichloride fomentations and anodynes, and enucleate as soon as all chance of saving the eye is gone.

DISEASES OF THE RETINA.

HYPERAEMIA AND ANAEMIA.

The retinal vessels do not participate much in the changes of the intra-cranial circulation. There is some retinal congestion in meningitis and always venous engorgement in papillitis and thrombosis of a retinal vein. The same condition of the veins, in milder form, is often met with in emphysema and in weakness of the heart's action.

Anaemia of the retina may result from embolism, from great loss of blood, from spasm of the arterial coats due to toxic doses of quinine and from spasm due to vaso-motor disturbance. The dimness of vision found in some cases of migraine or "blind headaches" are examples of the latter condition.

HYPERAESTHESIA AND ANAESTHESIA.

The former is manifested by photophobia, lachrymation and blepharospasm when exposed to ordinary sunlight. It may exist without any other lesion of the eye, and is generally due to hysteria, over use of eyes under bright light or neuralgia of the fifth nerve.

Anaesthesia of the retina is a rare condition characterized by fluctuations in acuteness of vision, contraction of the field, tendency of the eyes to tire quickly when used, inability to retain retinal images for any length of time and transient central scotomas or fading of objects from view when looked at closely. These symptoms are associated with a general neurasthenia, lack of mental energy, restlessness of body, depression of spirits, etc.

RETINAL CHANGE FROM DIRECT SUN RAYS; SNOW BLINDNESS: ELECTRIC OPHTHALMIA.

Persons who have looked directly at the sun have sometimes complained afterward of a central scotoma. These blind spots vary in their severity and persistency, sometimes being permanent. There may be central defect for colors, and also metamorphopsia. The ophthalmoscope will often show a minute lesion near the macula. The treatment consists of rest of eyes, dark glasses and hypodermatic injections of strychnia.

As a rule the only result, if any, of exposing the eyes to the glare of the snow is a mild form of conjunctivitis, but sometimes there is temporary, and in rare instances, permanent amblyopia.

Exposure of the eyes to strong electric light, as in electric welding, may result in severe changes, such as are found in injury by direct sunlight, and may take a mild form of ophthalmia, such as is occasioned by exposure to snow. Electric workers now prevent these conditions by using glasses deeply colored with yellow, ruby or a combination of deep blue and red.

EMBOLISM AND THROMBOSIS OF RETINAL VESSELS.

Symptoms of Embolism.—Sudden loss of vision, partial if the obstruction lodges in a branch artery, total if it stops in the main trunk. The blood vessels will be much reduced in size, the retina will be white and opaque, the macula appearing as a cherry red spot, owing to the fact that it is much thinner than the rest of the retina, and the choroid shows through it. The optic nerve generally atrophies. Vision is rarely restored.

Cause.—The plug may come from vegetations in the heart due to valvular disease or endocarditis. It may also result from aneurism of the aorta and from atheroma of the arteries. It also occurs with Bright's disease and pregnancy.

Symptoms of Thrombosis.—The extent of visual loss depends upon the location of the thrombus in the central vein or one of its branches. Vision is, as a rule, not lost as suddenly as in embolism. There will be oedema of the disc, tortuosity and engorgement of the veins, and numerous hemorrhages in the area drained by the thrombotic vein.

Cause.—Retarded venous circulation of the old, the emphysematous or those suffering with cardiac lesions. It may also be due to phlebitis.

Treatment of embolism and thrombosis is of little avail.

RETINITIS.

Inflammation of the retina may be limited to this membrane or may be associated with inflammation of the optic nerve (neuro-retinitis) or choroid (choroido-retinitis). The disc is usually involved unless the retinitis is very mild, and some opacity of the vitreous often co-exists. Normally the retina is a transparent membrane, but when inflamed it appears smoky or hazy and at times to such an extent as to obscure its vessels in some part of their course. The veins may appear unusually large and tortuous an

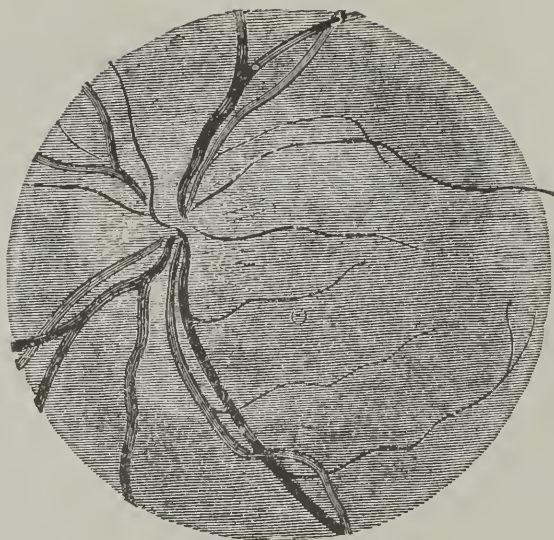


FIG. 72.—Serous retinitis. (Meyer.)

frequently there are hemorrhages. Often there will appear distinct white spots arranged around the macula or disc. These spots can be differentiated from choroidal atrophy by the absence of the pigmented border and the softness of their outline. In retinitis there will be dimness of vision in all degrees, perhaps limitation of the field, micropsia, megalopsia, metamorphopsia and night blindness. There will be no pain and no external sign of disease.

ALBUMINURIC RETINITIS.

Retinitis occurs in about seven per cent of all forms of albuminuria. It appears late in the stage of renal trouble, the majority of patients dying within two years after its advent. Both eyes are generally affected. The characteristic feature is the presence of the white spots of fatty degeneration above described. Complete recovery from albuminuric retinitis has been observed.

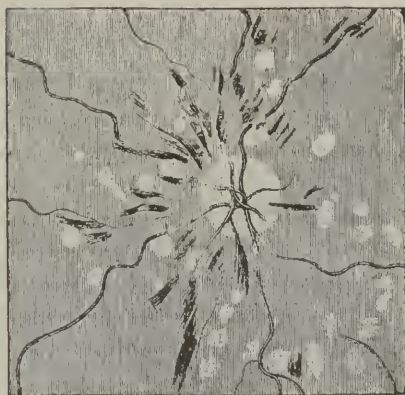


FIG. 73.—Recent severe retinitis in renal disease. (Gowers.)

SYPHILITIC RETINITIS.

This condition occurs from congenital as well as acquired syphilis. It is, as a rule, associated with choroiditis and opacity of the vitreous. Night blindness is a prominent symptom. The general characteristics are those enumerated above without the white spots, which are almost pathognomonic of renal disease.



FIG. 74.—Renal retinitis at a late stage. (Wecker and Jaeger.)

RETINITIS PIGMENTOSA.

This is a degenerative rather than an inflammatory condition. It is extremely chronic in its course, sometimes requiring years to reach its usual termination in blindness. Vision is much affected, but the symptom most complained of is nyctalopia (night blindness). The field of vision gradually contracts until only central vision is left. This much may remain for years. The fundus shows a peculiar stellate pigmentation beginning at its periphery and extending gradually to the macula. The calibre of the retinal vessels diminishes and there is slow atrophy of the retina and disc, with occasional opacity of the posterior lens capsule.



FIG. 75.—Pigmentary degeneration of the retina. (Jaeger).

The cause is obscure, but consanguinity of parents seems to be an etiological factor. No treatment is successful. Galvanism and strychnia have been recommended.

DETACHMENT OF THE RETINA.

This condition consists in a separation of the retina from the choroid, the intervening space being occupied by a serous fluid, blood or a tumor. Vision is affected in pro-

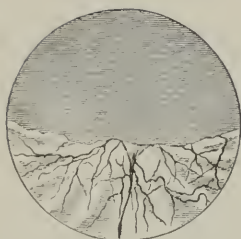


FIG. 76.—Ophthalmoscopic appearance of detached retina. (After Wecker and Jaeger.)

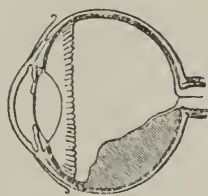


FIG. 77.—Section of eye with partial detachment of retina. (Nettleship.)

portion to the extent and location of the detachment, the field showing a defect corresponding to the position of the lesion. The ophthalmoscope reveals a steel gray reflex from the detached part, over which the retinal vessels flow. The presence of the vessels distinguishes this from any other condition presenting a similar reflex. If the detachment be recent there will be partial loss of vision, which increases with the degeneration of the retina. The retina rarely returns to its normal condition. The size of the detachment may remain stationary and may extend over the entire fundus.

Cause.—Blows upon the ball or jars by transmitted force. Tumors of the choroid. Disease resulting in fluidity or shrinkage of the vitreous. High degrees of myopia.

Treatment.—Long continued rest in bed with eye bandaged. Evacuation of the subretinal fluid by a knife, needle or pointed cautery; or absorption of it by pilocarpine sweats and abstinence from fluids. The results of treatment are discouraging.

CHAPTER XI.

DISEASES OF THE OPTIC NERVE AND ORBIT.

DISEASES OF THE OPTIC NERVE.

Optic Neuritis, or inflammation of the optic nerve may be divided into two kinds:

1. Papillitis, which involves the intra-ocular end of the nerve.
2. Retro-bulbar Neuritis, which affects the nerve between the ball and the chiasm.

PAPILLITIS.

The optic disc or papilla is the intra-ocular termination of the nerve or that part between the lamina cribrosa and the retina. With the ophthalmoscope the disc appears as a white, circular area in the orange colored groundwork of the choroid. The white reflex of the disc is caused by the lamina cribrosa showing through the transparent nerve fibres. In inflammation of the disc there are no definite signs. There is usually contraction of the field of vision and derangement of color perception, but vision may not be reduced until late in the progress of the disease. Main reliance in diagnosis is placed on the ophthalmoscope, which shows a serous infiltration of the disc manifested by redness, swelling and loss of its outline. The retinal arteries appear small and the veins filled and tortuous. The strangulation of the veins sometimes results in hemorrhages in the retina. In the great majority of cases both nerves are inflamed. If the inflammation extend by continuity of tissue to the retina, the condition is called neuro-retinitis. Papillitis may sometimes result in complete recovery, but in the majority of cases a partial or total atrophy of the nerve results.

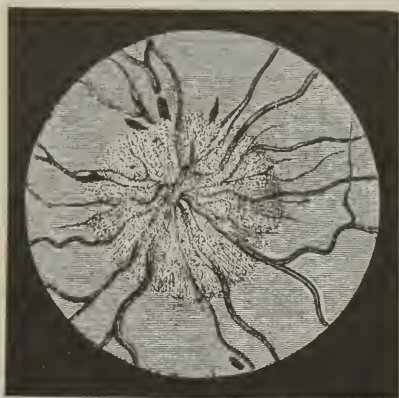


FIG. 78.—Appearance of a severe recent papillitis. Several elongated patches of blood near the border of the disc. (Jackson.)

Cause.—If monolateral it is generally the result of some orbital lesion. When bilateral it is usually due to intra-cranial disease, most frequently to tumors, but may be due to meningitis, abscess, depressed fracture or softening. It may also be the result of albuminuria, diabetes, syphilis, lead poison and anaemia. The prognosis will depend largely upon the etiology.

Treatment.—Forbid use of eyes. Direct your efforts to the cause, and when in doubt give iodide of potassium and build up the constitution by the usual methods.

RETRO-BULBAR NEURITIS.

This condition is also called central amblyopia and toxic amblyopia.

Symptoms.—Loss of acute vision, central scotoma for colors, and in advanced cases, central scotoma for objects. The affection is nearly always binocular and the vision of the two eyes nearly the same. In pronounced cases, the disc shows an unnatural whiteness of its temporal side, and in an advanced state the whole disc may present the appearance of atrophy. There is proliferation of connective

tissue in the nerve and atrophy of those fibers which go to the macula. The progress of the disease is slow, and the chance of recovery good unless of too long standing. It is almost exclusively a disease of men.

Cause.—It is due, in the great majority of cases, to the excessive use of tobacco or alcohol. Many observers consider tobacco the most potent etiological factor, and some entirely acquit alcohol of any responsibility for the disease. Exposure to cold, diabetes, rheumatism, syphilis and poison by some chemicals are supposed to be causative, and there are also some cases in which no cause can be discovered.

Treatment.—Absolute abstinence from the offending poison. Watch patient's digestion and give iodide of potassium or strychnine.

ATROPHY OF THE OPTIC NERVE.

In atrophy the medullary part of the fibres is displaced by granular fat and connective tissue, with thickening of the walls of the vessels and narrowing of their calibre.

Symptoms.—There is no pain and no change in the appearance of the ball, unless total blindness exists, when the pupils will be dilated. Great variety is manifested in the visual defects. Central vision is lowered and the field contracts, sometimes concentrically, sometimes irregularly. Color sense becomes defective or lost, perception of green, red, yellow and blue usually disappearing in the order named. If the atrophy be associated with spinal cord lesions we shall, as a rule, find the Argyll-Robertson pupil. The disc will appear abnormally white or gray, slight excavation due to shrinkage of its substance may be noticed and the retinal vessels will be reduced in size.

Cause.—Spinal diseases of which tabes dorsalis is the most important, papillitis, pressure of tumors, disease of the orbit, thrombosis and embolism of the retinal vessels, glaucoma, meningitis, syphilis, alcoholism, anaemia from

great loss of blood, and it may appear as a purely local disease independent of any other lesion.

Treatment.—Correct any derangement of general function. The galvanic current, one pole over the eye and the other at the back of the neck, is of doubtful utility. Give iodide of potassium, mercury or strychnia to the point of tolerance. Strychnia is more efficacious when given hypodermatically. Antipyrine, seven and a half grains every other day, hypodermatically is also recommended. Treatment is usually ineffectual.

DISEASES OF THE ORBITS.

From the position, structure and contents of the orbit we can readily conceive of some of the diseases which afflict it. We may have fractures of the bony walls; laceration of the soft parts by foreign bodies; emphysema caused by the escape of air from the lachrymal sac, the ethmoid cells or frontal sinuses; profuse hemorrhage from injury or straining; periostitis from injury, or secondary to other inflammation, etc. These are surgical conditions and can be diagnosed and treated on the lines of general surgery. There are two symptoms so constantly attendant upon orbital disease as to make them worthy of special mention.

1. Proptosis or exophthalmos.
2. Limitation of movement of the eyeball.

Associated with these salient symptoms are injection and chemosis of the conjunctiva, redness, swelling and oedema of the lids and severe pain, most noticeable when patients attempt to move the globe, or surgeon presses it backward into the orbit.

ORBITAL CELLULITIS.

This is an inflammation of the cellular tissue of the orbit.

Symptoms.—Proptosis with diplopia, pain, loss of movement of ball, injection and chemosis of the conjunctiva and

swelling and redness of the lids. As the severity of this disease varies greatly in different cases, we shall expect variation in the degree of manifestation of all symptoms. In the severe forms there will be chills with fever, and may be loss of vision due to pressure upon the optic nerve or disturbance of the intra-ocular circulation. There may be ulceration of the cornea and possibly suppuration of the whole eyeball.



FIG. 79,—Double orbital cellulitis, the result of erysipelas. (De Schweinitz.)

Cause.—The causes are such as produce cellulitis in other locations and are numerous. Special mention may be made of the severe form due to erysipelas, and also to the fact that it may arise by metastasis in all pyaemic conditions, especially puerperal septicaemia.

Treatment.—Support with tonics. Relieve pain by anodynes. Apply hot, one to five thousand bichloride fomentation, and as soon as any sign of fluctuation appears, open and treat antiseptically.

TUMORS OF THE ORBIT.

The orbit contains many different tissues, consequently a great variety of tumors may develop in this locality.

Exophthalmos and limitation of movement of the ball, without the usual manifestation of inflammation, are the most pronounced symptoms. Removal of the tumor is the treatment; without the ball, if the nature and extent of the growth will admit, with it if necessary.

CHAPTER XII.

FUNCTIONAL DISORDERS OF VISION, ETC.,

AMBLYOPIA AND AMAUROSIS.

These are terms used to express a diminution or loss of vision without any apparent lesion. The two words are used, more or less, indiscriminately but amaurosis is generally applied to the graver conditions. There are many forms of lowered vision, the pathology of which is known, but they do not come under this head. The cause of functional amblyopia may be known but the exact way in which the loss of vision is produced is unexplained.

1. A blow upon the eye or a traumatism to the head, direct or indirect, may be followed by amblyopia, more or less persistent. In these cases it is presumed that there is some invisible lesion of the parts concerned in vision.

2. Loss of blood may produce amblyopia, which probably is due to the fact that the retina is affected by the general lack of nutrition. But we do not know why the degree of amblyopia is not always commensurate with the amount of blood lost or why there is greater tendency to amblyopia from hemorrhages of the stomach, uterus and bowels than from traumatic hemorrhages.

3. Alcohol and tobacco have been mentioned as causes of retro-bulbar neuritis, in which there is pronounced lowering of vision. In addition to these agents amblyopia may be produced by a number of drugs, among which are nitrate of silver, mercury, salicylic acid, cannabis indica, chloral, lead and quinine. De Schweinitz has seen a decided temporary loss of vision from twelve grains of the last named drug. Quinine is supposed to produce amblyopia by its action on the vaso-motor centers, thus constricting the vessels and diminishing the peripheral blood supply.

4. Amblyopia, without any ophthalmoscopic change, is occasionally associated with diabetes. We know that retinal hemorrhages are often the result of diabetes, and we presume that in these cases which show no lesion of the fundus, that there are hemorrhages in the optic nerves.

Amblyopia is sometimes found in uremic poison, especially the uremias of pregnancy and scarlatina. The prognosis as to sight in these cases is good.

Temporary partial or total loss of vision is also, at times, due to chronic malarial poison.

5. Hysterical amblyopia may occur in both sexes but is most frequent in females. As might be supposed the symptoms assume a great variety of forms, such as total blindness, hemianopsia, scotoma, color blindness and contraction of the visual field. Neurasthenic school children, especially girls, are frequently thus afflicted and great tact and judgment are required in their treatment.

HEMIANOPSIA.

Hemianopsia and hemianopia are terms used to express loss of one half of the visual field. Both eyes are usually affected, which indicates a lesion in or back of the chiasm. If only one eye is affected the lesion is in front of the chiasm. In the great majority of cases of hemianopsia affecting both eyes the diminution or loss of vision will be in the right half of each field or the left half of each field. This is called homonymous hemianopsia. We may have loss of the external half of each field called temporal hemianopsia, or of the internal half of each field called nasal hemianopsia but these conditions are rare. A condition still more uncommon is loss of the upper or lower field. The lesion which causes hemianopsia may lie at any part of the visual tract from the eye ball to the cortex of the brain and a knowledge of the origin and distribution of the optic nerve fibres is necessary to determine its location. The lesion may be a tumor, periostitis, blood clot, soften-

ing of the brain, atheroma of adjacent vessels, injuries, etc. Treatment must be directed to the cause.

NYCTALOPIA OR NIGHT BLINDNESS.

Most authors use the word hemeralopia to express night blindness and nyctalopia to express day blindness. Greenhill has proven that they are in error and that the reverse is correct according to derivation and ancient usage. The night blindness of retinitis pigmentosa must not be confounded with the functional variety being described in which there are no visible lesions of the fundus. In functional night blindness vision may be normal in a bright light but is greatly lowered on dull days, in the twilight or in dimly lighted rooms. It is found in persons who have been exposed to glaring light, such as travelers in the tropics, glass blowers, electric welders and those who work before furnaces. It is also supposed to be associated with certain states of lowered vitality such as scorbutus, starvation, etc.

The treatment consists in protection of the eyes by dark glasses, the use of tonics of quinine, iron, strychnia and cod liver oil, and changing the occupation if that seems at fault.

HEMERALOPIA OR DAY BLINDNESS.

This condition is the opposite to nyctalopia in that the patient sees better and greatly prefers diminished illumination. It is found as a symptom in retro-bulbar neuritis, albinism, dilatation of the pupil from third nerve, paralysis or from the use of a mydriatic, central cataract, etc. It also exists as a functional condition independent of any demonstrable lesion. Functional hemeralopia is found in persons who have been excluded from the light for a long period and is also a frequent symptom of hysteria.

When hemeralopia is a symptom, the primary affection should be treated. If it is functional examine closely for muscular insufficiency and errors of refraction; build up

the constitution and accustom the eyes to light by resorting to the strategy demanded by each case.

MALINGERING.

Patients will pretend to be blind from various motives. It may be due to a desire to exaggerate an injury over which a law suit is pending, to secure a pension, to escape some disagreeable duty or to excite sympathy. For obvious reasons, but one eye is claimed to be affected. Numerous tests will reveal the patient's hypocrisy, if he claims blindness in but one eye, among which are the following:

1. Put on him a pair of spectacles, one lens of which is plain glass and the other a prism with its base up or down. If malingering he will see double and an effort to walk, especially to go down stairs, will be made so cautiously that his true condition is detected.

2. Place before the eye he claims is bad a plain glass and before the other a plus glass just strong enough to obscure its vision. If with these, vision is normal, the patient is malingering.

3. Hang in a window at a convenient distance some letters of green glass. Hold before the good eye a glass colored red. If he reads the letters, he does it with his bad eye as the green letters can not be seen through the red glass.

If the patient claims to be blind in both eyes his detection is more difficult and a close watch may be necessary to determine the true condition. A simple test, which may be of service, is to ask the patient to look at his own hand. A blind man will turn the eyeballs toward the hand, a malingerer may intentionally look in some other direction.

DIAGNOSTIC TABLE.

	CONJUNCTIVITIS.	INTERSTITIAL KERATITIS.	IRITIS.	ACUTE GLAUCOMA.
Redness.	Palpebral and ocular conjunctiva injected. Pericorneal zone the last part to get red.	Pericorneal zone injected. In bad cases the entire ocular conjunctiva becomes red.	Pericorneal zone injected first, entire ocular conjunctiva liable to become congested.	Diffuse redness with conjunctival chemosis and oedema of the lid.
Pain.	Burning and scratchy as there was a foreign body in eye.	Generally slight, but in some cases severe.	Sometimes absent but usually severe, following the fifth nerve into the cheek and forehead.	Always severe, following the fifth nerve into the cheek and forehead.
Vision.	Unimpaired or slightly diminished by mucous on the cornea.	Greatly diminished and same in all parts of the field of vision.	Diminished by turbidity of the aqueous humor but not as bad as in interstitial keratitis or acute glaucoma.	Greatly diminished; nasal side of field first and most.
Discharge.	Muco-purulent or purulent.	Watery.	Watery and profuse.	Watery.

Pupil.	Normal.	Normal.	Contracted and sluggish.	Dilated and of a green color.
Tenderness on pressure.	None.	None.	Slight. Considerable over the ciliary body if it be involved.	Great.
Tension.	Normal.	Normal.	Possibly slightly increased.	Greatly increased.
Temperature, pulse, etc.	Normal.	Normal.	Normal.	Increase in temperature and pulse, sometimes vomiting.
Cornea.	Normal.	Hazy, pink or buff colored sometimes hypersensitive.	Normal.	Hazy, with sub-normal sensitiveness.
Age of patient.	Any.	Disease of childhood, rarely seen after 25 years.	Any age, but rare before puberty.	Rarely seen in patients under 35.

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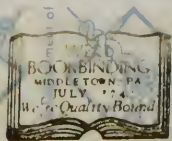
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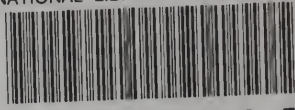
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